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CARCINOMA OF THE COLON

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MEMPHIS, TENNESSEE

Carcinoma of the colon differs from cancer in other areas of the body in one significant respect: It offers a better prognosis. As in most malignancies, the prognosis depends primarily upon the stage at which the disease is discovered. It is often difficult to make an early diagnosis, since the symptoms and signs are by no means peculiar to carcinoma of the colon. They may, in fact, be far more suggestive of some other disease. In such cases, the fate of the patient may depend upon the clinician's "cancer consciousness." As a rule, however, the diagnosis may be readily suspected if one bears in mind that the right and left halves of the colon differ in anatomy and function, and that carcinoma presents certain definite and different clinical manifestations in the two halves.

The right colon is large and movable; its walls are thin and permeable, its contents are liquid, and its function is digestion and absorption. Carcinomas in this half, especially below the hepatic flexure, are usually large, bulky, ulcerative growths of the medullary or mucoid type, with irregular, extensive surfaces. Because of the large lumen, the liquid contents, and the tendency of the malignant growth to perforate and form an abscess, obstruction is seldom pronounced.

The left colon is smaller than the right; it is more tubular and muscular, and, with the exception of the sigmoid, is immovable. The function of the left colon is storage, and its contents are soft or solid. Malignant growths on this side are of a scirrhous or fibrous character, encircling the bowel and producing obstruction early in their course. The majority are located in the sigmoid flexure.

Growths in the transverse colon may be either proliferative or constrictive in type. Those near the hepatic flexure are more often proliferative, whereas those near the splenic flexure are usually constrictive.

Diagnosis

Carcinoma of the right colon

On the whole, carcinoma of the right colon manifests itself later and presents more diagnostic difficulty than cancer of the left half. The outstanding symptoms produced by lesions on the right are: anemia, with loss of appetite, weight and strength; digestive disturbance or a sense of discomfort in the abdomen; and a palpable mass in the right side or middle of the abdomen. In addition, the rapid absorption from extensive ulcerating growths in this location usually gives rise to toxic symptoms, such as fatigue, nervousness, insomnia, and headache. Pain and melena usually indicate a late stage of carcinoma in the right colon.

Anemia has been observed in almost two thirds of our patients with carcinoma of the right colon. Weakness and loss of appetite were associated in approximately 50 per cent of these. Not infrequently, the sole clue to the diagnosis was the patient's complaint of profound weakness, suggesting the anemia. In many cases the resemblance to pernicious anemia was striking; in fact, the distinction was not always clear until the roentgenogram revealed the malignancy.

Digestive symptoms—excessive gas, nausea and vomiting, and diarrhea, frequently

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alternating with constipation—have been reported by more than half of our patients with carcinoma of the right colon. Blood was found in the stools of half the patients. Practically the entire group complained of discomfort, soreness, or some degree of pain in the epigastrium or right side of the abdomen. Pain, especially if severe and intermittent, was almost universally associated with definite obstructive symptoms, such as gaseous distention, borborygmus, and visible peristalsis. In carcinoma of the right colon, these symptoms most often point to obstruction at the ileocecal valve or the hepatic flexure, and occasionally to volvulus or intussusception incident to the tumor.

The main diagnostic difficulty offered by many carcinomas of the right colon characterized by digestive disturbances and pain is their distinction from gallbladder disease, appendicitis, duodenal ulcer, and amebiasis. This distinction is particularly difficult if the symptoms are not clearly defined. In view of the prevalence of the latter diseases, it is but natural that the clinician should think of them first. He should also think of carcinoma, however, in every patient who presents a digestive complaint, and should not consider the examination complete until the possibility has been excluded or confirmed by roentgenographic studies. The more vague the symptoms referable to the right colon, the more suspicious should one be of carcinoma.

A palpable mass has been observed in more than half of our patients with carcinoma of the right colon. Several of the patients had discovered the mass themselves and had come for examination solely on this account. In such cases also, a roentgenogram is imperative to exclude the possibility of other types of lesions and to confirm the presence of a carcinoma.

Carcinoma of the transverse colon

The symptoms and signs of carcinomas of the transverse colon depend upon their type and location—whether they are proliferative or constrictive, and whether they are in the right or left half of the segment. In the right half they are more likely to exhibit the characteristics of right-colon malignancy; beyond the middle, they generally present those of left-colon lesions.

Carcinoma of the left colon

The outstanding feature of carcinoma of the left colon is obstruction. As a rule, this manifests itself insidiously, as an alteration of bowel function—either constipation or diarrhea, or both alternately. More often than not, blood and mucus appear in the stool sooner or later. Loss of appetite, nausea, and vomiting are not uncommon. Pain is experienced earlier than with lesions of the right colon; it may be dull and aching, though generally it is intermittent and cramp-like. A palpable, tender, firm mass may be elicited, especially if the growth is in the transverse segment or sigmoid. Visible or palpable peristalsis and a rumbling, gurgling or tympanitic sound, with abdominal distention relieved by the passage of gas, complete the picture. Not infrequently, the location of the growth may be indicated by the fact that borborygmus and peristalsis cease at that point. Urinary infection incident to pressure of the tumor upon the urethra may produce urinary frequency, rigors, and fever. A sense of weight in the lower abdomen and pain in the back and down the legs are other symptoms which are often associated. Anemia, weight loss, and cachexia are usually late manifestations of carcinoma of the left colon.

In our experience, pain was the most common symptom of carcinoma of the left colon, being present in more than three fourths of the cases. In almost three fourths of this number the pain was severe. Two thirds of the patients reported constipation. Distention was present in more than half the cases, and bloody stools in almost half. Approximately a fourth of the patients had diarrhea, nausea and vomiting, and borborygmus. It is noteworthy that a third of the patients were anemic.

Carcinoma of the rectum

The diagnosis of carcinoma of the rectum should be relatively easy. The outstanding features are constipation, a sense of fullness and pressure within the rectum, frequently pain and tenesmus on defecation, and the passage of bright blood and mucus. Other prominent symptoms and signs are abdominal distention, pain, a mild diarrhea, anemia, weight loss, and weakness. In these cases also, urinary disturbances incident to pressure upon the urethra may be described. As

a rule, a mass can be felt in the rectum on digital examination, and can be seen through the proctoscope. Almost 90 per cent of our patients with rectal carcinoma had observed blood in the stools, and more than 50 per cent complained of constipation, rectal discomfort, and rectal or abdominal pain of greater or less degree. The growth was palpable in 83 per cent of the cases, and in the remainder was apparent on proctoscopic examination.

Digital examination and proctoscopy should be an integral part of the study of any patient who presents symptoms referable to the lower colon and rectum. Proctoscopy is useful not only for visualizing lesions beyond the palpating finger, but likewise for taking specimens for microscopic study. Biopsy should be made whenever an ulcerating or bleeding rectal mass is found. Too often a malignant growth in the rectum is mistaken for some other disease, usually hemorrhoids. Eleven per cent of our patients with carcinoma of the rectum had been treated for bleeding hemorrhoids.

The importance of x-ray examination

Not infrequently, the diagnosis of carcinoma of the colon may be made from the history alone, and one should always be able to elicit one or more symptoms to suggest its presence. We have had a few cases in which the history provided the sole clue to the diagnosis, the entire examination being negative. As a rule, however, the diagnosis hinges upon the roentgenogram. Every patient who presents a single symptom of carcinoma which has persisted over a period of several weeks should have a complete roentgenographic study of the gastro-intestinal tract, unless the cause is found on digital and proctoscopic examination. The gastrointestinal series should include studies following the introduction of barium not only by mouth, but by enema; fluoroscopic examination should be employed during injection of the barium to reveal any lesions which might be obscured by overlapping areas of the colon.

Surgical Treatment

The notable improvement in the morbidity and mortality associated with surgery of the colon within recent years may be attributed in large part to improved methods of preoperative and postoperative care. We have come to realize the advantages of preparing these patients so that they have every opportunity of coming through the surgical procedure safely and, with the help of the new postoperative measures, of going on to recovery. Such patients are usually in a poor nutritional state as a result of their inability to take food; they are dehydrated, anemic and toxemic, and their blood chemistry values are materially altered. Fortunately, even the most desperately ill may now be prepared so that operation may be performed with reasonable safety. Modern preoperative treatment consists in the administration of fluids intravenously; blood transfusions; a high-calorie, high-vitamin, and low-residue diet; the use of amino acids, blood plasma and serum globulin to restore protein deficiencies; and sulfonamide therapy to minimize the danger of infection.

Carcinoma of the right colon

For carcinoma of the right colon, we consider the one-stage operation with ileotransverse-colostomy the best procedure. Unless obstruction is severe, it is unnecessary to use the Miller-Abbott tube or make a vent to the surface to overcome distention. In more advanced obstructions, the use of the tube for a few days prior to operation will often decompress the bowel sufficiently to permit the performance of a right colectomy in one stage.

The choice of the type of anastomosis in right colectomy is largely a matter of individual preference. Formerly, we employed the end-to-side technique (end of the ileum to the side of the transverse colon), but in more recent years have preferred the side-to-side anastomosis using an open method. The wound is usually closed without drainage, and satisfactory bowel function is quickly established. Experience has proved that there is little danger of peritonitis and wound infection.

Until a few years ago, surgeons everywhere emphasized the importance of complete and thorough peritonization of the gutter after removal of the right colon. The majority have now abandoned this painstaking part of the operation, no longer making an effort to cover the raw space left by the resection. Our own results have been equally as good without peritonization as with it.

In the literature, frequent references are made to the use of the Mikulicz operation for right colectomy. We have never felt justi-

fied in subjecting patients to multiple operations when a one-stage procedure can be performed so safely. With the latter, the patient is well and out of the hospital within two or three weeks, whereas with the Mikulicz technique the total hospitalization covers a period of several weeks. Thus, not only does the Mikulicz procedure necessitate repeated surgical risks and an unnecessarily prolonged period of disability, but it also imposes a greater financial burden on the patient. If one could prove the two-stage operation to be safer, the elements of time and expense might be disregarded; but our experience has not shown that it affords any greater measure of safety.

Carcinoma of the transverse colon

For carcinoma of the transverse colon, we prefer a one-stage resection with end-to-end anastomosis. The majority of our patients with carcinoma in this segment have had only a mild degree of obstruction and have not required a preliminary decompression operation. The Mikulicz operation is satisfactory for growths of the transverse colon, but again has the disadvantage of involving multiple stages, with their attendant risks and prolonged period of hospitalization.

Carcinoma of the left colon

For lesions of the splenic flexure and left colon we formerly employed the Mikulicz principle almost exclusively; during the past several years, however, we have performed primary resection routinely. A large wedge of the gland-bearing mesocolon may be removed with equal ease by either method. In several cases we have employed primary resection, with end-to-end anastomosis, for lesions low in the sigmoid.

If the obstruction is of mild degree, we do not make a vent to the surface. In our early experience with primary resection and end-to-end anastomosis, a temporary tube colostomy was done, with the idea of avoiding postoperative distention and leakage at the suture line. This practice was discontinued some time ago, and, thus far, we have not observed evidence of leakage in a single case. If the obstruction is severe, however, a preliminary transverse colostomy is made and the distal segment is irrigated for two or three weeks before the resection is done; the colostomy is closed later. If the colon is mobilized so as to permit closure without tension, if the different layers of the colon walls are sutured properly, with adequate closure of the mesocolon and complete coverage of raw space, and if sulfonamide drugs are given prophylactically, obstruction and infection are remote possibilities. The ultimate success or failure of the operation obviously depends upon whether all the malignant tissue, including the glands, is removed.

Carcinoma of the rectum

During the past several years we have performed an abdominoperineal resection for almost all carcinomas of the rectum, and for a number of cancers at the recto-sigmoid juncture. In all except our earlier cases, the operation has been completed in one stage.

We are of the opinion that the first consideration in the treatment of carcinoma of the rectum and rectosigmoid is to cure the patient. This being true, one should not permit sentiment to deter him from doing a thorough resection, and removing the anus. We have performed a number of anterior resections for lesions in the rectosigmoid or upper rectum, leaving the anus. This procedure, however, has been limited to cases in which there was extension to neighboring structures and possible metastases in the liver. It is a palliative measure, designed only to remove the growth, overcome obstruction, and make the patient more comfortable.

One feature of the surgical technique which we unfailingly observe concerns the closure of colostomy wounds. We have found that delayed closure—that is, packing the wound with antiseptic gauze, allowing the sutures to remain untied, and, forty-eight hours later, removing the gauze and tying the sutures tight—is the best protection against wound infection. Primary healing takes place in practically every case.

The importance of exploration and radical extirpation

Our experience with colon surgery has taught us that the abdomen should always be explored throughout at the time of laparotomy. Occasionally, one will be surprised to find multiple malignancies, either in the colon alone or associated with growths elsewhere. We have had this experience in several of our cases.

With all the refinements of preoperative and postoperative care, we have grown more and more bold in our surgical attack upon carcinoma of the colon. We are now doing resections even in the presence of a moderate amount of liver metastases, having found that occasionally a life is thereby prolonged a surprising length of time, and that the patient's remaining existence is at least made more tolerable by extirpation of the original growth.

We do not hesitate to do a resection when the lesion has invaded an adjacent viscus, provided the risk is not too great. If one follows the rule of doing as radical an extirpation as is possible, he may sometimes add years to a patient's life.

Types of Operations and Distribution of the Lesions

In our experience, more than three fourths of all diseases of the colon requiring operation have been carcinomas. This statement does not include patients who had merely an exploration nor those whose condition was regarded as hopeless without exploration. We have explored or operated upon a total of 233 patients. Of these, 32 had an exploration, 28 had a palliative operation, 4 had a local excision of a malignant polyp, and 170 had a resection. One patient had two resections at an interval of ten years, making a total of 234 operations. The distribution of the lesions is shown in table 1.

Table 1

Distribution of Carcinomas of the Colon in 234 Cases

Location	*No.*	*Per Cent*
Right colon	37	15.7
Transverse colon	19	8.5
Left colon	101	43.2
Rectum	77	32.7

Results of Operation

Since, for purposes of this discussion, we are interested only in resections, we have analyzed the operative and ultimate results in those cases in which this procedure was employed. In order to evaluate better the newer methods of preoperative and postoperative treatment, as well as the newer surgical techniques, we have divided the cases into two groups: (I) those in which resection was performed before 1939, and (II) those operated on after that date. The number of resections and the operative mortality are shown in table 2.

Table 2

Resections of the Colon and Rectum for Carcinomas in 234 Cases (233 Patients)

Group	*Cases*	*Resections*		*Operative Deaths*	
		No.	*Per Cent*	*No.*	*Per Cent*
I (Prior to 1939)	59	35	58	10	28
II (1939-1947)	175	135	77	5	3.7
Total	**234**	**170**	**72.6**	**15**	**9**

It will be observed that, in our experience, the mortality associated with resections for carcinomas of the colon has decreased approximately 24 per cent since 1939. Furthermore, better preparation of patients and the use of better methods of anesthesia enabled us to perform resections in a large number of poor-risk patients. Another factor responsible for the higher percentage of resections in group II is the extension of our criteria for resection.

Operative mortality

Of the 5 postoperative deaths in group II, 4 followed abdominoperineal resection for rectal carcinoma, giving a mortality rate of 8.3 per cent in the 48 cases of rectal carcinomas in this group. One of these patients, a man aged 68 years, should not have had an operation, as he was a diabetic and suffered from cardiovascular disease. He apparently died of either a cerebral embolus or an acute heart attack. One, a 45-year-old woman, died on the table without any apparent cause. The third, a woman aged 60, died of acute urinary retention; and the fourth, a man of 74, died of peritonitis following acute distention and perforation of the sigmoid.

The one remaining death in group II followed a two-stage resection of the sigmoid with end-to-end anastomosis, and was due to uremia. The patient was a man 75 years of age. This one death constitutes an operative mortality of 2 per cent for resections of the left colon and 0.87 per cent for all resections of the colon, exclusive of the rectum, since 1939 (table 3).

Table 3

Mortality of Resections for Carcinomas of the Colon and Rectum

Group	*Rectum*	*Deaths*		*Colon*	*Deaths*	
		No.	*Per Cent*		*No.*	*Per Cent*
I (Prior to 1939)	10	1	10	25	9	36
II (1939-1947)	48	4	8.3	87	1	0.87
Total	**58**	**5**	**8.6**	**112**	**10**	**9**

We have had no fatalities from colon resections in more than six years, since we began to employ one-stage resections of the transverse and left colon.

Survival rate

Of 60 patients who had a resection for carcinoma of the colon or rectum more than five years ago, 1 is living twenty-four years later, 5 are living fifteen or more years later, 4 have survived ten years or longer, and 13 have survived five years or more. These 23 patients represent 38.3 per cent of the 60 on whom resection was performed. During this same period 33 patients had either a palliative operation or merely an exploration. On the same percentage basis, 12 of these patients would have lived five years or more had they come to operation before their condition had become hopeless. This figure does not take into consideration those who did not even have an exploration.

Our percentage of ten-year survivals is considerably higher than our five-year survival rate. This fact would indicate that our efforts to bring these patients to operation while their disease is still curable have been of little avail. On the other hand, the fact that 38.3 per cent of the patients in this group who had the benefit of resection have survived from five to twenty-four years illustrates the favorable prognosis that may be offered the unfortunate victims of carcinoma of the colon when the lesion is recognized and removed before the malignant cells have spread beyond the reach of the scalpel.

The training of the doctor, which began with observations on and the care of the sick individual, is due now for a great forward stride. Observations on whole communities, whether great or small (or on appropriate samples) and improved health provisions for them, must henceforward become the prior objective. The individual is not likely to suffer neglect in the process, for all communities are composed of individuals. For generations yet we shall doubtless continue to build our costly hospitals and clinics, and require our armies of practitioners and ancillaries, but meanwhile we must at least embark upon the crusade which will end in the steady reduction of waiting-lists and the closure of hospital wards, and which will eventually put the physical, mental and moral health of peoples before their material wealth. In that crusade—whether by our researches, by realistic reforms in teaching, by the better education of the people or direct representations to government—it is our first duty as physicians to explore and prepare the way.—John A. Ryle: Social Pathology and the New Era in Medicine, Bull. New York Acad. Med. 23:329 (June) 1947.

STREPTOMYCIN IN TULAREMIC PNEUMONIA

With Two Case Reports

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Although there have been many recent reports emphasizing the remarkable efficacy of streptomycin therapy in tularemic infections[1], we feel that presentation of the following cases is justified in order to stress the importance of early diagnosis of the pleuropulmonary form and to point out a relatively frequent complication, pleural effusion.

Case Reports

Case 1

A 40-year-old white male salesman was admitted to the Charlotte Memorial Hospital on January 2, 1947, complaining of severe weakness and malaise of five days' duration. Early one morning, as he was hunting, he felt overpowering weakness and malaise. Next day his temperature rose to 102 F., and thereafter varied between 102 and 103 F. until admission. Headache developed two days before admission, and he was "out of his head off and on" thenceforth. Nausea with occasional vomiting had been present three days. There were no symptoms referable to the chest except a slight, nonproductive cough. The patient had been hunting rabbits daily for at least two weeks before the onset of his illness, and skinning each day's bag barehanded.

Physical examination upon admission revealed an acutely ill, restless white male. The temperature was 100 F., pulse 120, respiration 24. The blood pressure was 114 systolic, 70 diastolic. Except for occasional sibilant rales over the lung bases and tenderness to deep palpation beneath the right costal margin, there were no significant findings.

Admission laboratory data were as follows: red blood cells, 4,360,000; hemoglobin, 13 Gm. (90 per cent); white blood cells, 6,550, with 89 per cent neutrophils, 10 per cent lymphocytes, and 1 per cent eosinophils. Urinalysis was normal except for a trace of albumin. A roentgenogram of the chest made on the second hospital day showed pneumonia in the left infraclavicular area and in the lateral aspect of the right chest.

1. (a) Heilman, F. R.: Streptomycin in Treatment of Experimental Tularemia, Proc. Staff Meet., Mayo Clin. 19: 553-559 (Nov. 29) 1944.
(b) Foshay, L., and Pasternack, A. B.: Streptomycin Treatment of Tularemia, J.A.M.A. 130:393-398 (Feb. 16) 1946.
(c) Gordon, A. M.: Streptomycin in Tularemia, J.A.M.A. 132:21-22 (Sept. 7) 1946.
(d) Howe, C., Correll, L. L., Borkwalter, H. L., and Ellington, H. V.: Streptomycin in Tularemia, J.A.M.A. 132:195-200 (Sept. 28) 1946.
(e) Foshay, L.: A Comparative Study of the Treatment of Tularemia with Immune Serum, Hyperimmune Serum, and Streptomycin, Am. J. Med. 1:180-188 (Aug.) 1946.
(f) Hartnett, W. G., and Mollica, S. G.: Tularemia: Treatment of a Case with Streptomycin, South. M. J. 39:774-777 (Oct.) 1946.
(g) Nichols, D. R., and Herrell, W. E.: Streptomycin: Its Clinical Uses and Limitations, J.A.M.A. 132:200-206 (Sept. 28) 1946.
(h) National Research Council, Chester S. Keefer, M.D., Chairman: Streptomycin in Infections, J.A.M.A. 132: 70-77 (Sept. 14) 1946.
(i) Hunt, J. S.: Pleuropulmonary Tularemia, Ann. Int. Med. 26:263-276 (Feb.) 1947.

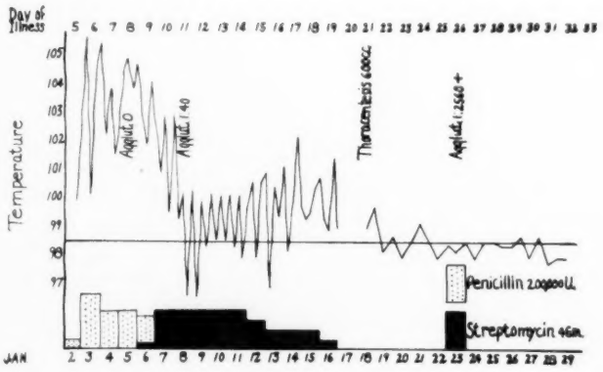

Fig. 1. Note prompt response of the temperature curve to streptomycin, yet failure to reach normal levels until after thoracentesis. The temperature was not charted on January 17.

The patient's course during the next four days was alarming. On the second hospital day he had a shaking chill followed by a rise of temperature to 105.8 F. (fig. 1). He was irrational and delirious most of the time. The cough became more harassing, and drenching sweats were frequent. Nausea and vomiting occurred once or twice daily. Though the presumptive diagnosis upon admission was atypical pneumonia, penicillin was administered as a precautionary measure. As a glance at figure 1 shows, it had no beneficial effect.

Streptomycin was started on the fifth hospital day in doses of 0.5 Gm. (500,000 units) intramuscularly every three hours. Within twenty-four hours the patient was no longer irrational, and his fever had begun to fluctuate at lower levels. During the next nine days, he held his clinical gains and noted an increase in appetite, while his temperature, still swinging in type, remained at much lower levels. He continued to cough, however, without raising significant amounts of sputum, and to complain of an almost constant aching pain in the right lower chest anteriorly, accentuated by coughing. On the sixteenth hospital day a friction rub was heard over the right base posteriorly and in the axilla. Next day flatness to percussion, absence of tactile fremitus and breath sounds, and egophony at the seventh right interspace posteriorly denoted pleural effusion. Thoracentesis produced 600 cc. of serosanguineous, almost coffee-colored fluid with a specific gravity of 1.016 which, on smear, showed numerous red blood cells and white blood cells. Thereafter, the patient rapidly improved. His temperature remained normal, he complained of no chest pain or cough, and he began to regain weight and strength. Discharged on the twenty-eighth hospital day as much improved, he continued to do well; six weeks after dismissal he had reached normal weight, after having lost 25 pounds at the peak of his illness.

Changes in the agglutination level were pronounced (fig. 1). Agglutination tests with Pasteurella tularense were negative at first, but the titer rose to 1:40 on the third day of streptomycin therapy and to 1:2560+ fifteen days later, after 30.5 Gm. of streptomycin had been given over an eleven-day period. X-rays showed progression in the pneumonic process from admission to the thirteenth hospital day (fig. 2), but had markedly improved two days before discharge (fig. 3).

Comment

The diagnosis in this case was by no means obvious. Symptoms of severe intoxication — high, relatively sustained fever, drenching sweats, delirium, nausea, and weakness—were much more prominent than local symptoms of cough and chest pain. Physical examination, too, yielded meager information. The history of contact with rabbits in a gravely ill patient with roentgeno-

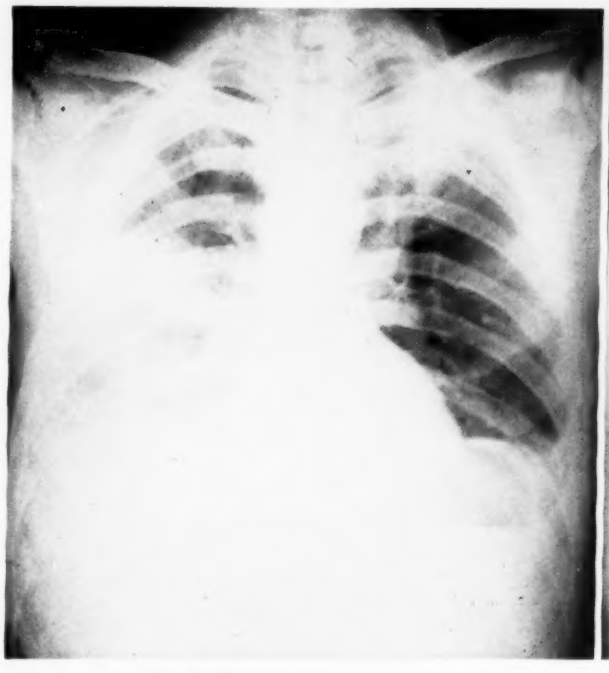

Fig. 2. Chest film taken on seventeenth day of illness, showing maximal spread of pneumonia.

Fig. 3. Chest film taken on thirtieth day of illness, showing pronounced clearing of right lung and left apex, though the diaphragm is still elevated at the site of fluid removal.

graphic evidence of atypical pneumonia led to the use of streptomycin. Clinical improvement was rapid and dramatic; in our opinion, the patient owes his life to the drug. Complete recovery, however, did not ensue. The persistence of dry cough, chest pain, and low-grade fever was doubtless due to a pleural reaction which eventually resulted in the formation of pleural fluid, serosanguineous in type. When this was withdrawn, recovery was unimpeded.

A high agglutination titer quickly developed, rising from zero to 1:2560+ in two and a half weeks. Although roentgenograms showed a spread of pulmonary infiltration from January 3 to January 14 (two days before streptomycin was discontinued), resolution of the process was rapid thereafter, as the film of January 27 shows. The amount of streptomycin used was greater than that said to be necessary to control tularemia, but we felt that the desperate condition of the patient warranted maximal dosage.

Case 2

A 10-year-old farm boy was admitted to Good Samaritan Hospital on June 25, 1947, in a state of mental confusion, disorientation, and drowsiness. Five days before, he had noticed general malaise, followed next day by shaking chills, nausea, vomiting, and drowsiness. Vomiting continued through the next day. High fever was noted on the second day and persisted until admission. Throughout the illness, high fever, drowsiness, and mental confusion were the most outstanding symptoms. There were no complaints referable to the chest. Five days before the onset of his illness, the patient caught and played with a young wild rabbit.

Outstanding findings on physical examination were a temperature of 104 F., with a pulse rate of only 100. Except for pronounced drowsiness and dryness of the skin, there were no other remarkable findings.

Emergency laboratory studies showed a red cell count of 4,660,000, a hemoglobin of 82 per cent, and a white cell count of 10,100.

A presumptive diagnosis of typhoidal tularemia was made, and streptomycin therapy (3 Gm. every twenty-four hours in divided doses) was instituted. On the second day (fig. 4) there was a sharp drop in temperature, but confusion, disorientation, and hallucinations persisted. Despite the absence of chest signs, an x-ray made on the fourth hospital day showed pneumonia extending from the right hilum (fig. 5). By the fifth hospital day, all mental symptoms had disappeared, and the patient began eating well. On the sixth hospital day the temperature rose to 101.8 F., and the dosage of streptomycin, which had been dropped to 1 Gm. a day, was raised to 1½ Gm. a day. Next day the temperature spiked to 103.5 F. Since there were no further physical signs to be observed, a presumptive diagnosis of drug fever was made and streptomycin was discontinued, a total of 13½ Gm. having been administered. Thereafter, the patient's course was entirely normal, and he was discharged as cured on the fifteenth hospital day.

The rapid resolution of pneumonia, which reached its greatest extent on the seventh hospital day

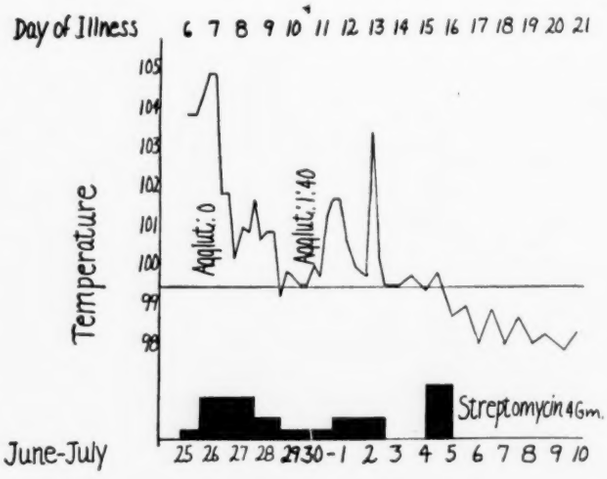

Fig. 4. Note the prompt response to streptomycin initially, followed by a febrile reaction on the two days before the drug was discontinued. All temperatures were taken rectally.

(fig. 6), is seen by the final film taken on the fourteenth hospital day (fig. 7). On the seventh hospital day, physical signs of consolidation were pronounced on the right posteriorly from the fifth interspace downward. These signs had begun to disappear by the ninth day, and at discharge the chest was clear to percussion and auscultation. Agglutination tests with Pasteurella tularense, negative on the second hospital day, had become positive in a dilution of 1:40 by the sixth hospital day. A third agglutination test was made on the fourteenth hospital day, but has not as yet been reported.

Discussion

Diagnosis

Except in the typhoidal type, which comprises about 6.4 per cent of all cases[2], the diagnosis of tularemia is seldom difficult. A history of exposure to rabbits, or, less commonly, to squirrels, opossums, weasels, or ticks[3] arouses strong suspicion. Within a few hours to two weeks after exposure (two to five days on the average[3b]) general symptoms of severe illness develop, often suddenly, including chills, fever, headache, aching, nausea and vomiting, and cough. About forty-eight hours later the primary lesion, skin or conjunctival, and associated lymphadenopathy become apparent[3b]. Differentiation into clinical types has been standardized for years; Foshay[3b], in reviewing 600 cases of tularemia, found 87.5 per cent to be of the ulcero-glandular type, 3 per cent of the glandular type, 1.8 per cent of the oculo-glandular type, and 7.7 per cent of the typhoidal type.

This last group, most important for our

2. Stuart, B. M., and Pullen, R. L.: Tularemic Pneumonia, Am. J. M. Sc. 210:223-236 (Aug.) 1945.
3. (a) Blackford, S. D., and Casey, C. J.: Pleuropulmonary Tularemia, Arch. Int. Med. 67:43-71 (Jan.) 1941.
(b) Foshay, L.: Tularemia, Medicine 19:1-83 (Feb.) 1940.

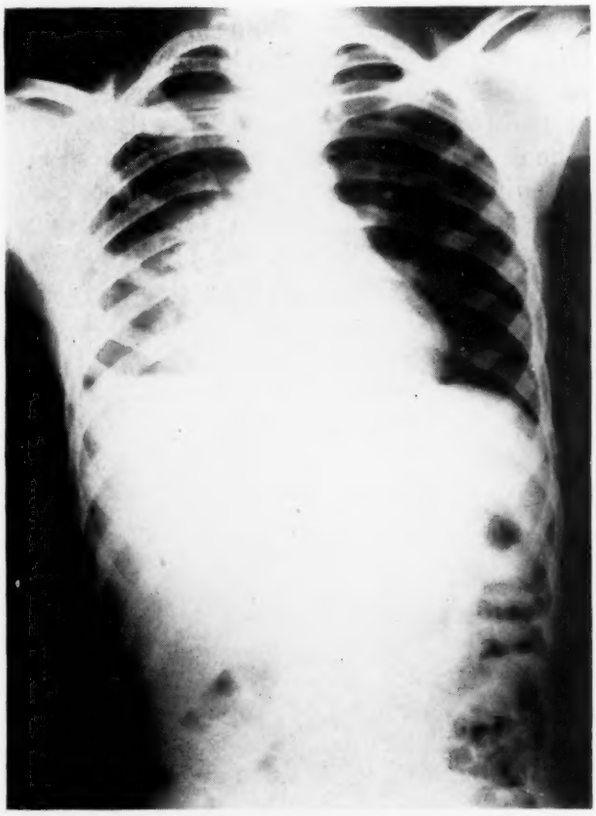

Fig. 5. Chest film taken June 28, showing an early spread of pneumonia from the right hilum.

present consideration, shows no primary lesion and no lymphadenopathy[3b,4]. Only the general symptoms listed above, and the additional grave signs of prostration, delirium, stupor, and coma may be observed. The agglutination test, never positive in the first week and sometimes negative in the third or even the fourth week[3b], offers little help in the early stages of the disease. Indeed, laboratory personnel who possessed agglutinins as a result of receiving Foshay serum showed a sharp drop in agglutination titer after acquiring the disease[14]. Pneumonia occurs with far greater frequency in the typhoidal type than in other types[2,3]. More than a third (36.2 per cent) of all cases of tularemic pneumonia reported in the American literature[2] were of the typhoidal type. A large number of cases of tularemic pneumonia, therefore, will show no diagnostic features. Roentgenographic signs are those of atypical pneumonia, sometimes with later consolidation of one or more lobes[2,5]; Bihss and Berland[5] found that in typhoidal tularemia

4. Scott, J. W., and Macbeth, R. A. L.: Tularaemia, Canad. M. A. J. 55:564-566 (Dec.) 1946.
5. Bihss, F. E. and Berland, H. I.: Roentgenological Manifestations of Pleuropulmonary Involvement in Tularemia, Radiology 41:431-457 (Nov.) 1943.

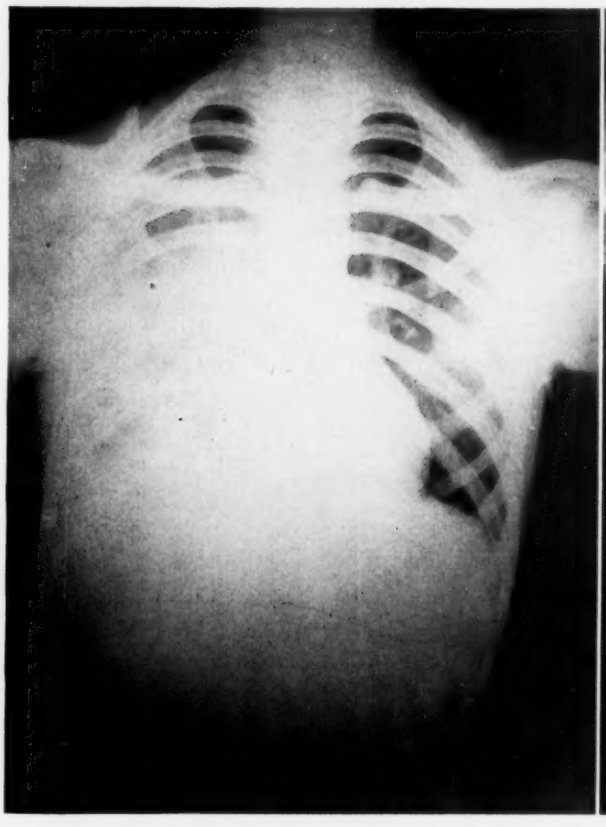

Fig. 6. Chest film taken July 1, showing maximal pneumonic involvement of the right middle and lower lobes.

Fig. 7. Final film taken July 8, showing pronounced clearing except for a small area about the right hilum.

hilar lymphadenopathy, striking in other types of the disease, was not an early feature. In the absence, then, of definite diagnostic criteria elicitable by physical examination, x-ray studies, or serologic methods, a trial of streptomycin as advocated by Hunt[1i] should be given each patient with severe atypical pneumonia.

Treatment

The treatment of tularemia has, since the advent of streptomycin, become at the same time simple and highly effective. Stimulated by the report of Heilman concerning the dramatic effect of streptomycin on tularemia in mice[1a], many investigators and clinicians have described similar effects in human cases[1b-i]. Of 67 cases reported to the National Research Council[1e], 63 recovered. In two of the failures the diagnosis was questionable and the total dosage of streptomycin grossly inadequate; in these and one other, treatment was begun two months after the onset of symptoms. One patient with pleuropulmonary tularemia died on the first day of treatment; 10 recovered. Subsequently Hunt[1i] has reported dramatic improvement in 11 out of 12 cases of pleuropulmonary tularemia; the one patient who died had cerebral complications. Thus, a disease carrying an overall mortality rate of 6 per cent[3b], and a rate in the pleuropulmonary form of 39.9 per cent[2], has been brought almost entirely under control by the use of streptomycin.

The details of the administration of streptomycin have been related elsewhere[1g-i]. It is injected intramuscularly in a concentration of 100-200 mg. per cubic centimeter every three to six hours; the daily dosage usually recommended for tularemia is 1 to 2 Gm. In severe pleuropulmonary cases, or in the typhoidal type when the patient is showing signs of septicemia, larger doses may be given. As a rule administration is discontinued after seven days, but may be prolonged in severe cases.

Although local pain at the site of injection—a discomfort somewhat relieved by mixing procaine into the diluting fluid—is frequent, other toxic effects of streptomycin are not often observed. These include[6]: the "histamine-like" reaction, with headache, flushing, and nausea; the "sensitization" type, with fever and a nonspecific skin eruption; and the "neurological" type characterized by vertigo, tinnitus, and deafness. This last manifestation necessitates withdrawal of the drug; sensitization may be reduced by shortening the interval between injections. In the dosage recommended for tularemia, however, severe reactions are unlikely.

It must be emphasized that streptomycin should be started as soon as possible—certainly within the first fifteen days of the disease[1e]. Where doubt as to the diagnosis exists, as in pleuropulmonary disease without a demonstrable primary lesion, a therapeutic trial of 2 Gm. daily for two days is strongly recommended[1i].

Complications

Factors delaying recovery include pneumonic lesions and their sequelae, liquefaction necrosis of lymph nodes, and lesions of serous membranes[1e]. Our immediate concern is with pleural effusion. Although in 1941 Blackford and Casey[3a] were able to find only 30 cases of hydrothorax in all the literature on tularemia, Stuart and Pullen[2] later reported some pleural reaction in 50 per cent of all cases of tularemic pneumonia. In 3 of Hunt's 12 cases[1i] serosanguineous fluid was aspirated from the pleural cavity. Streptomycin does little to prevent fluid formation. Thoracentesis was necessary in Hunt's cases within four to nineteen days after streptomycin had been started.

The first case in the present report (see figure 1) graphically shows the need of watchfulness for the common complication of hydrothorax. While streptomycin caused rapid improvement in the patient's general condition and temperature curve, complete recovery did not ensue until after thoracentesis.

Resolution of the pulmonary exudate is somewhat accelerated by streptomycin, but x-ray signs may persist long after clinical improvement has become established. Pleural effusion appears to prolong complete healing and should be anticipated.

Summary

1. Two cases of severe pleuropulmonary tularemia of the typhoidal type, successfully treated with streptomycin, are reported.

2. The difficulty of exact diagnosis in the early stages of the disease is emphasized, and

6. National Research Council, Chester S. Keefer, M.D., Chairman: Streptomycin in Infections, J.A.M.A. 132:4-11 (Sept. 7) 1946.

a therapeutic trial of streptomycin in similar cases of severe atypical pneumonia is strongly urged.

3. The incidence and management of a common complication of tularemic pneumonia tending to delay recovery—pleural effusion—are briefly discussed.

211 Hawthorne Lane

AN EVALUATION OF THE USE OF ANTIBIOTICS IN DERMATOLOGIC DISEASES

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So many clinical reports have been published on the use of antibiotics in dermatology that it is difficult to evaluate their true worth. This confusion is to be expected when a new medicament becomes widely available. It is probably true that the sulfonamide series of drugs are not clearly evaluated even today, after some ten years of widespread use; it was only a few months ago that an authoritative article suggested the abandonment of sulfanilamide dusting in wounds. Many new pharmaceutical preparations which have too short a period of laboratory investigation cause an initial burst of enthusiasm in clinical trial, then a period of condemnation and abandonment; many months or years later a proper appraisal of the drug's real worth and place in medical treatment is made. Perhaps such a course of events can be avoided with regard to the antibiotics, but at this early stage it is too soon to be sure, despite the fact that scientists have known something about antibiotics for over fifty years, and have experimented with at least forty-two different kinds.

The most widely used antibiotic, penicillin, has been readily available for something less than three years, and during this period it has been used in many types of skin diseases. Neglect of the fundamental principles of dermatologic treatment—such as the absorption of discharges from oozing lesions, the debridement of all encrusted lesions prior to the application of topical remedies, the use of the proper vehicles which will penetrate or mix with serous discharges, or soften tough and horny crusts—and of the surgical principle of establishment of drainage in lesions which enclose pus, has resulted in conflicting reports which are confusing to all concerned.

It is well to answer, if possible, the following questions before using one of the antibiotics in any dermatologic condition.

1. Is the disease due to a microbial organism, and is that organism susceptible to the antibiotic?

2. If the answer to the first question is in the affirmative, then one should ask: Can the lesion be reached without further measures, such as removal of crusts and debris, or surgical drainage?

3. The third question should be: What vehicle should be used in order to get the medicament to the lesion? Should it be applied as an aqueous compress, given parenterally, or used in a water-soluble, absorptive-base ointment, or possibly in a water suspension?

The above questions automatically go through the dermatologist's mind as he considers the problem before him, and in the evaluation of the antibiotics these principles must be kept in mind. An effort to compare the effectiveness of penicillin, streptomycin, and tyrothricin with other methods of treatment seems the most practical approach to this evaluation, with particular reference to (1) the time needed to effect a cure, (2) the relative efficacy of the treatment when applied by an untrained person (the patient), and (3) the ability of the particular agent to effect a satisfactory outcome in a disease heretofore resistant to other methods of treatment.

Penicillin

With the above criteria in mind, four tables have been prepared to show the results which may be expected with penicillin in various dermatologic conditions. Table 1 lists the dermatoses in which penicillin appears to be superior to other treatments. Of these ten diseases, only one, impetigo contagiosa, responds readily to topically applied penicillin, preferably an ointment consisting of 500 units of penicillin per gram of water-soluble ointment base. Pyogenic dermatitis may respond to penicillin compresses containing 250 units per cubic centimeter of normal saline, but it is advisable to use parenteral penicillin as well, since the disease generally involves the deeper layers of the skin

Read at a Symposium for Medical Alumni, Duke University, Durham, April 24-26, 1947.

Table 1[1]

Anticipate Excellent Results with Penicillin in

- **Impetigo Contagiosa**
- **Cellulitis, Acute**
- **Pyogenic Dermatitis**
- **Anthrax**
- **Vincent's Stomatitis**
- **Syphilis, Cutaneous**
- **Yaws, Cutaneous**
- **Gangrenous Stomatitis (noma)**
- **Erysipelas**
- **Erysipeloid (swine bacillus)**

also. In all the other conditions listed, penicillin should be given intramuscularly in doses of not less than 25,000 units of the aqueous solution every three hours for five to seven days, or 300,000 units of the peanut oil-beeswax emulsion daily for five to seven days. Occasionally a longer period of treatment will be necessary, depending upon various local factors, and in the case of cutaneous syphilis a much higher total dose given over a proportionately longer time will be required.

Table 2[1a,b,2]

Anticipate Good Results with Penicillin in

- **Ecthyma (ulcerative impetigo)**
- **Impetigo Neonatorum**
- **Carbunculosis**
- **Cellulitis, Chronic**
- **Actinomycosis**
- **Tropical Ulcer (phagedenic)**
- **Hidroadenitis Suppurativa (chronic axillary adenitis)**
- **Infectious Eczematoid Dermatitis**
- **Nodular Non-Suppurative Panniculitis (Weber-Christian disease)**
- **Bullous Orificial Erythema Multiforme (Stevens-Johnson disease)**

Table 2 lists ten conditions which respond as well to penicillin as to any other known means, and the choice of therapy may rest upon factors of economy, ease of application in the home, or any other reasonable basis. For some of the diseases listed in this category penicillin may eventually be the treatment of choice. Inasmuch as very few patients with Weber-Christian or Stevens-Johnson disease are seen, the reports are not yet fully confirmed, and it is considered advisable to place these conditions in the "good result" rather than in the "excellent result" group.

Table 3[1c,3]

Anticipate Fair Results with Penicillin in

- **Furunculosis**
- **Sycosis Vulgaris (barber's itch)**
- **Pemphigus Vulgaris (?)**
- **Lupus Erythematosus, Chronic Discoid (?)**

Table 3 concerns five diseases in which penicillin is of doubtful value. Furunculosis responds temporarily in most instances, but a few cases apparently are influenced favorably and do not recur. Note the inclusion of pemphigus vulgaris in this table. It is one of the diseases generally regarded as not responsive to penicillin, but in 2 reported cases —one from Duke Hospital—pemphigus has been relieved temporarily, at least, by penicillin. In this almost universally fatal disease, any aid is well worth mentioning.

Table 4[1c]

Penicillin Has Not Proved of Value in

- **Acne Vulgaris**
- **Ulcer, Hemostatic**
- **Ulcer, Decubitus**
- **Dermatitis, Hemostatic**
- **Dermatitis Repens**
- **Lupus Erythematosus, Acute Disseminate**
- **Coccidioidomycosis, Cutaneous, Late**
- **Lymphopathia Venereum**
- **Chancroid**
- **Granuloma Inguinale**
- **Tuberculosis, Cutaneous**
- **Leishmaniasis Americana**
- **Blastomycosis, Cutaneous**
- **Dermatitis Herpetiformis**
- **Moniliasis (thrush)**

Table 4 is a list of fourteen dermatoses in which penicillin is of no value, or in which another remedy is far superior.

Streptomycin

Experience with streptomycin has been limited because of the small supply. Only five conditions are listed in table 5 as being responsive to penicillin, and in none of these has a sufficient number of cases been observed over a long enough period to prove unequivocally streptomycin's value. Time and an increasing supply of the drug will tell us much in the future. Results in cutaneous tuberculosis have been good in some cases, but relapses in scrofuloderma have been observed several times. The antibiotic must be given parenterally in doses of 1 to

1. (a) Pillsbury, D. M.: The Management of Bacterial Infections of the Skin, J.A.M.A. 132:692-698 (Nov. 23) 1946.
   (b) Wright, C. S. and Gross, E. R.: Topical Treatment with Penicillin Ointment, Arch. Dermat. & Syph. 55: 52-56 (Jan.) 1947.
   (c) Neuroth, M. L. et al.: A Study of Penicillin Ointments and Creams, J. Am. Pharm. A. 35:321-324 (Nov.) 1946.
2. (a) Aldrich, C. A. and Holmes, C. A.: Treatment of Impetigo Neonatorum with Minimal Doses of Penicillin, Am. J. Dis. Child. 72:279 (Sept.) 1946.
   (b) Goldfarb, A. A.: A Case of Stevens-Johnson Disease Treated with Penicillin, J. Pediat. 28:579-589 (May) 1946.
3. Callaway, J. L., Arena, J. M., Noojin, R. O. and Riley, K. A.: Pemphigus, Successful Treatment with Penicillin, J. Pediat: 28:592-594 (May) 1946.

Table 5[4]

Streptomycin Is of Apparent Value in
Tuberculosis, Cutaneous
Chancroid
Dermatoses due to Proteus Vulgaris
Dermatoses due to other gram-negative organisms
Granuloma Inguinale

Streptomycin Is Not of Value in Syphilis, Cutaneous

4 Gm. daily, and over a period of sixty to ninety days. The present cost of the drug makes it prohibitive for all but very wealthy patients unless it is available under research grants.

The use of streptomycin in the other dermatoses listed has been reported only in isolated cases or is still in the experimental stage. Streptomycin has not proven of value in the control of rabbit syphilis, and there is no indication that it will be of use in human syphilis.

Tyrothricin

Tyrothricin, a mixture of gramicidin (25 per cent) and tyrocidin (60 per cent), is an antibiotic suitable for local use only. It is commonly used as a compress in an aqueous-alcoholic solution containing 100 mg. of tyrothricin per 100 cc. of liquid, or as "intraderm" tyrothricin, a specially prepared penetrating wetting agent. It is insoluble in saline solution and does not appear to be particularly effective in ordinary ointment bases.

Tyrothricin has proven useful in a number of cutaneous dermatoses of infectious origin (table 6). Its chief value seems to be in its lack of sensitizing properties, but its performance has not been dramatic as a rule. It may well be used when difficulties are encountered with penicillin or other proven types of medication, especially in chronic ulcerative conditions requiring prolonged treatment.

Table 6[5]

Tyrothrycin May Be of Help in
Furunculosis
Ecthyma
Folliculitis
Decubitus Ulcer
Acne Vulgaris Pustulosa
Hemostatic Ulcer
Impetigo Contagiosa
Cellulitis, Acute and Chronic
Sycosis Vulgaris

Tyrothrycin Has Not Been Helpful in Dermatitis Repens

Table 7[1c,6]

Penicillin

1. **Modes of administration**
   - **a) Intramuscular**
   - **b) Intravenous**
   - **c) Topical**
2. **Toxic manifestations**
   - **a) Upon parenteral or oral administration**
     - **Urticaria**
     - **Erythema multiforme**
     - **Exfoliative dermatitis**
     - **"Serum sickness" reaction**
     - **Erythema**
   - **b) Upon topical use**
     - **Contact sensitization**

Table 8[4,7]

Streptomycin

1. **Modes of administration**
   - **a) Intramuscular**
   - **b) Intravenous**
   - **c) Topical**
   - **d) Oral use unsatisfactory**
2. **Toxic manifestations**
   - **a) Upon parenteral administration**
     - **Tinnitus (often severe)**
     - **Vertigo (often severe)**
     - **Deafness (may be permanent)**
     - **Various vesicular, papular, and erythematous eruptions**
   - **b) Upon topical use**
     - **None known**

Table 9

Tyrothrycin

1. **Mode of administration**
   Topical only, as this substance is too toxic for other use
2. **Toxic manifestations**
   Few encountered thus far. Any substance may act as a sensitizer, however

Problems for the Future

As may be noted, only forty-odd skin conditions are listed in all the tables. There are more than three hundred well-defined entities in dermatology, and a large number of these are due to infectious agents, but it will

4. (a) Hinshaw, H. C., Feldman, W. H., and Pfuetze, K. H.: Treatment of Tuberculosis with Streptomycin, J.A.M.A. 132:778-782 (Nov. 30) 1946.
(b) O'Leary, P. A., Ceder, E. T., Hinshaw, H. C. and Feldman, W. H.: Treatment of Various Types of Cutaneous Tuberculosis with Promizole and Streptomycin, Arch. Dermat. & Syph. 55:222-232 (Feb.) 1947.
(c) Herrell, W. E., and Nichols, D. R.: The Clinical Use of Streptomycin. Proc. Staff Meet., Mayo Clinic 20: 449-462 (Nov. 28) 1945.
5. Rankin, L. M.: The Use of Tyrothrycin in the Treatment of Ulcers of the Skin, Am. J. Surg. 65:391-392 (Sept.) 1944.
6. (a) Dressler, S. and Dwork, R. E.: Reactions to Penicillin, J.A.M.A. 133:849 (Mar. 22) 1947.
(b) Vogel, H. R.: Reactions to Penicillin, Arch. Dermat. & Syph. 54:713 (Dec.) 1946.
(c) Pillsbury, D. M., Steiger, H. P. and Gibson, T. E.: The Management of Urticaria Due to Penicillin, J.A.M.A. 133:1255-1258 (April 26) 1947.
(d) Callaway, J. L. and Barefoot, S. W.: Immunological Studies on Patients Developing Urticaria Associated with Penicillin Therapy, J. Invest. Dermat. 7:285 (Dec.) 1946.
7. (a) Fowler, E. P., Jr. and Seligman, E.: Otic Complications of Streptomycin Therapy, J.A.M.A. 133: 87-91 (Jan. 11) 1947.
(b) Kecfer, C. S.: Streptomycin in Infections, J.A.M.A. 132:70-77 (Sept. 14) 1946.

require a much longer period of observation to obtain an evaluation of the usefulness of antibiotics in all of these dermatoses. Of the forty-odd discussed here, only three conditions are without any promise of cure—acute disseminated lupus erythematosus, dermatitis herpetiformis, and late cutaneous coccidioidomycosis. Perhaps some of the recently studied antibiotics, such as bacitracin, may offer hope for victims of these diseases in the future[8].

Reasons for failure

The following reasons may be listed for some of the failures with antibiotic therapy: (1) Not all bacteria and other infective agents are inhibited by our presently available antibiotics. (2) Apparently it is necessary to use them in an intensive fashion at the beginning of therapy, since there is considerable evidence that resistance to these agents may be induced when small initial doses are used. (3) Local and general hypersensitivity to penicillin develops in a relatively high percentage of cases (5 per cent or more) when it is used for more than a few days. (4) Due emphasis has not been placed upon the fact that the physician must make it possible for the antibiotic to reach the focus to be treated. (5) The antibiotics do not of themselves alter the individual's immunobiologic relationship with the underlying pathologic condition.

If these reasons for failure were generally recognized and the antibiotics were not expected to do all the work alone, our treatment results would undoubtedly improve. A recent surgical article states: "No available anti-bacterial agent can completely sterilize an open wound. Topical or local chemotherapy has been discarded as ineffective and deleterious."[9] The present discussion would lend support to this conclusion, although it is probable that topical therapy with antibiotics is not as often deleterious as it is ineffective.

Toxic reactions (tables 7, 8, and 9)

The relatively high incidence of toxic reactions to penicillin and streptomycin is a major drawback. The addition of a generalized urticaria, a simple erythema, or erythema multiforme to a relatively benign dermatosis may be startling to the patient, and is often very upsetting. An exfoliative dermatitis or a serum-sickness type of reaction, with painful swelling of the joints, may prove so serious as to prolong illness considerably, or to hospitalize a previously ambulatory patient. One of the anti-histamine drugs, benadryl or pyribenzamine, does help to alleviate most of these side-reactions, and may, if given concurrently, prevent their appearance. Occasionally, however, it will be necessary to stop the medicament because of the potential danger associated with a generalized exfoliative form of dermatitis, or a serum-sickness reaction. The vertigo and permanent deafness associated with the administration of streptomycin are very serious drawbacks to its use; in tuberculosis, however, the seriousness of the disease must be weighed against the dangers of the therapeutic agent.

Summary

1. Penicillin is of great value in the treatment of certain microbial dermatoses due to penicillin-susceptible organisms.

2. Streptomycin may prove of help in cutaneous tuberculosis and dermatoses due to gram-negative organisms.

3. Tyrothricin is of value in chronic ulcerative dermatoses and in other skin conditions when prolonged medication is indicated.

4. Adherence to sound principles of dermatologic debridement and effective surgical drainage, and observance of the limitations of the antibiotics are necessary for satisfactory results in dermatologic therapy.

5. The untoward reactions to penicillin and streptomycin may be serious enough to necessitate discontinuance of therapy.

6. Suitable vehicles for the antibiotics and simultaneous parenteral administration in large dosage are prerequisites for successful topical therapy.

7. The propensity of penicillin to induce local and general hypersensitivity is a serious objection to its prolonged or repeated topical use.

811 South Twentieth Street

8. Meleney, F. L., and Johnson, B.: Bacitracin Therapy, J.A.M.A. 133:675-680 (Mar. 8) 1947.
9. Lyons, C.: Chemotherapy in the Management of Wounds, J.A.M.A. 133:215 (Jan. 25) 1947.

Security versus liberty.—Growing numbers of people are stressing security rather than liberty. Even when lip service is paid to the old ideals of freedom, many individuals show by their actions an increasing readiness to sacrifice limitations on organized power for the sake of security.—Evan W. Thomas: The Increasing Complexities of Medical Care, New York Medicine 3:22 (June 5) 1947.

CONGENITAL ANOMALIES OF THE SPINE AND BACKACHE

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The study of congenital anomalies of the spine in relation to backache is the study of structural weakness in relation to stress and strain.

The basic plan of nature for the vertebral column was that of a horizontal weight-bearing mechanism. As man's position has become more erect, the increase in the vertical weight-bearing function of the spine has not been matched by corresponding changes in the physical and mechanical make-up of that structure.

All body weight above the pelvis is now carried by the lumbosacral articulation. It is at this point that the need for efficacy of design and integrity of structures is greatest. We find here, however, a joint poorly adapted to weight bearing, and one in which the percentage of congenital anomalies is greater than at any other point.

The articular plane of the first sacral vertebra is normally 15 to 20 degrees below horizontal; this angle creates an incline down which the fifth lumbar vertebra would slide, were it not for the restraint offered by the articular facets, the spinal and sacrospinal ligaments, and the sacrospinalis muscle. The normal curvature of this part of the spine partially distributes this strain throughout the lumbar vertebral structures, but the burden of maintaining its integrity is borne by the ligaments, muscles, and articulations of the lumbosacral area.

The usual congenital anomalies found in this region are, in order of frequency, as follows:

1. **Faulty fusion (spina bifida, manifest or occult)**
2. **Asymmetric articular facets**
3. **Transitional vertebrae**
   - **(a) Sacralization of the fifth lumbar vertebra —unilateral or bilateral, with or without synarthrosis**
   - **(b) Lumbarization of the first sacral vertebra —unilateral or bilateral**
4. **Prespondylolisthesis of the fourth or fifth lumbar vertebra, or both**
5. **Spondylolisthesis—the fourth on the fifth lumbar vertebra, the fifth lumbar vertebra on the first sacral vertebra, or (rarely) the first on the second sacral vertebra.**

Certain other anomalies may be added to this group, although they involve structures other than the lumbo-sacral spine:

1. **Wedged vertebra of the dorsal area**
   - **Transverse (hemivertebra), producing scoliosis**
   - **Anteroposterior, producing kyphosis**
2. **Other congenital anomalies producing postural abnormality**
   - **Pelvic anomalies**
   - **Difference in leg length**
   - **Clubfoot and other pedal anomalies**
   - **Paralysis from a birth injury, affecting the trunk or extremities.**

Read before the Catawba Valley Medical Society, Lenoir, February 18, 1947.

Fig. 1. Incomplete fusion of the first and second sacral vertebrae, together with asymmetric facets throughout the lower lumbar spine.

Fig. 2. Bilateral sacralization of the fifth lumbar vertebra.

Fig. 3. Unilateral sacralization of the fifth lumbar vertebra, with the formation of pseudoarthrosis. Note arthritic changes in the pseudoarthrosis.

Fig. 4. Bilateral lumbarization of the first sacral vertebra.

Fig. 5. Unilateral complete lumbarization of the first sacral vertebra, with pseudo-arthrosis of the opposite, partially lumbarized segment, and arthritic changes in this structure.

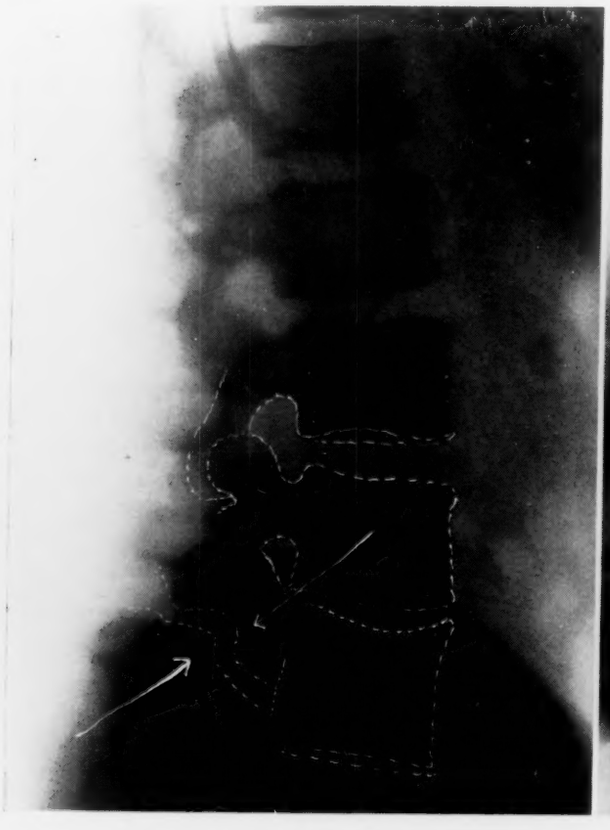

Fig. 6. Defect of continuity of the posterior neural arch of the fifth lumbar vertebra, without displacement (prespondylolisthesis).

Fig. 7. Anteroposterior view of bilateral defect of continuity in the pars interarticularis.

Faulty fusion is inevitably associated with faulty ligamentous and muscular development. When the integrity of the neural arch is absent, the attachments of ligaments and muscles are correspondingly reduced; the local area is weaker, and a greater strain on structures above and below that level is produced. Such an area is more easily subject to strain or sprain.

Asymmetric facets limit the excursion of an articulation, and by so doing place greater demand upon contiguous structures. They also create torsional strain, the effects of which may be transmitted to other levels.

Sacralization of the fifth lumbar vertebra, if bilateral, produces complete immobilization of the lumbosacral joint, thus demanding greater mobility of the remaining lumbar structures, and placing a greater strain upon them. If the process is unilateral, marked torsional strain may be added to the burden of the superjacent structures.

Bilateral lumbarization of the first sacral vertebra, for all practical considerations, adds another segment to the lumbar spine, decreasing the structural solidarity of that area, and increasing strain upon structures governing its functions. Since the sacro-iliac articulations are reduced by one segment, the functions of the lumbo-sacral joint are transferred, to a certain extent, to the articulation of the first and second sacral vertebrae. Inasmuch as neither the ligamentous attachments nor the intervertebral structures of this joint were designed for this function, this area suffers from the effects of such added strain.

As in the case of sacralization, unilateral lumbarization adds torsional strain to the burden of the first sacral vertebra and superjacent segments.

In *prespondylolisthesis*, the defect of continuity of the posterior neural arch is bridged by a fibrocartilaginous material. In this condition maximum function of the muscles and ligaments must be constantly maintained to prevent the separation of the weakened area, and the consequent forward-slipping which may occur (spondylolisthesis).

Fig. 8. Posterior neural arch defect of the fifth lumbar vertebra, with questionable slight forward displacement of the fifth lumbar vertebra on the first sacral vertebra (prespondylolisthesis? spondylolisthesis?).

Fig. 9. Marked defect in continuity of the posterior neural arch of the fifth lumbar vertebra. Slight forward displacement of the fifth lumbar vertebra and the first sacral vertebra is noted (spondylolisthesis).

In *spondylolisthesis,* the mechanical disturbance produced by the slipped vertebra is added to the strain of supporting the weakened area.

Whenever either prespondylolisthesis or spondylolisthesis of the articulation of the first and second sacral vertebrae occurs coincident with lumbarization of the first sacral vertebra, the mechanical disturbance is markedly exaggerated. Such a combination is, however, relatively rare.

A *wedged vertebra,* through the production of either scoliosis or kyphosis, may produce postural strain of other segment levels; it may also produce torsional strain, if the wedging be in the oblique plane.

Developmental anomalies of the pelvis, lower extremities, dorsal, or cervical region may create low-back strain through the postural situations they produce. Low-back pain may thus be due to a tilted pelvis, a spinal list, a shoulder droop, or a torticollis as readily as to an anomaly of the lumbosacral region.

Routine roentgenologic studies prove that many congenital anomalies of the spine may exist asymptomatically. Whenever backache and spinal anomaly co-exist, however, the probable causal relationship between the structural defect and the clinical picture must be considered.

Summary

Congenital anomalies of the lumbosacral spine may be responsible for weakness and strain of the lower back.

Certain other congenital anomalies may contribute to low-back strain through the postural situations they produce.

The structural weakness represented by congenital spinal anomaly may be a predisposing factor in the development of clinical symptoms.

Allowing the patient to talk. Although time consuming it is important to allow the patient to tell his story with as few interruptions as possible because in this way important associations are obtained and the ground work laid for a good relationship with the patient. Occasionally the patient has to be led back to a discussion of the present illness, but aside from such interruptions it is best to allow him to tell the story in his own way. Then one may go over the history in chronologic order with questions regarding the very first evidences of illness.—Edward Weiss: Psychosomatic Diagnosis; Am. Practitioner 1:160 (Nov.) 1946.

MILK SICKNESS

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Milk sickness is an intoxication in human beings which is caused by the ingestion of milk and milk products of cows which have eaten white snakeroot (Eupatorium urticaefolium) or rayless goldenrod (Aplopappus heterophyllus). It may be acute or subacute, and probably may become chronic or relapsing. The primary intoxication in animals is known as "trembles" or "split-tail."

Hardin's report in 1934 reviewed his experience with milk sickness at this hospital[1]. Since this report was published, 21 additional cases have been seen here. Between 1941 and 1946 only one patient with acute milk sickness was admitted, but in the past year we have seen 3 cases of this disease, with one fatality.

Case Reports

Case 1

W. L. P., a white farmer 53 years of age, entered Grace Hospital on August 18, 1946. During the preceding five weeks he had suffered from severe and persistent nausea, loss of appetite, weakness, and obstinate constipation. The presence of white snakeroot on his farm was denied; milk sickness had not occurred there for at least fifty years. No other member of his family was suffering from similar symptoms, but it was noted that he alone had a decided predilection for cream and butter.

Some days prior to admission barium studies of the stomach and gastric analysis were performed in the hospital clinic, with negative findings. The diagnosis of milk sickness was then made, and the ingestion of brandy and honey to the point of intoxication was prescribed. Three days of such treatment at home was unavailing because of violent emesis and increasing prostration. The patient was then brought to the hospital.

The outstanding physical findings on admission were paroxysmal unproductive retching, considerable dehydration, and alcoholic intoxication. Routine laboratory determinations were normal. Large quantities of brandy were given by gavage, and infusions of glucose were given by vein. Treatment was discontinued on the third hospital day, and he was able to tolerate a liquid diet without vomiting. He was discharged on August 23, 1946. At last report, six months later, he remains weak, listless, without appetite, and unable to engage in strenuous work.

Case 2

H. J., a 3-year-old white boy, was brought in coma to Grace Hospital on November 8, 1946. For three days he had been languid and without appetite, and for two days he had vomited copiously. Shortly

Read before the Avery County Medical Society, April 25, 1947.

1. Hardin, R. H.: Milk Sickness, Virginia M. Monthly 61: 528-531 (Dec.) 1934.

before admission he had a generalized clonic convulsion, followed by stercoraceous vomiting and coma. When he was admitted, the reflexes were absent. He was cyanotic, and was obviously suffering from acute pulmonary edema. Intranasal oxygen was given with no benefit, and he expired before further studies could be performed.

Permission for autopsy was refused, and the diagnosis was entirely speculative. Shortly afterwards, however, Dr. B. B. McGuire, county health officer, reported that he had visited the child's home and had found an abundant growth of white snakeroot on the farm. He was fully satisfied that the death had been due to milk sickness. This case has been previously reported[2].

Case 3

N. J., an 11-year-old sister of the deceased child, was brought to Grace Hospital on November 12, 1946, because of loss of appetite and vomiting for three days. The mother and a sister were likewise affected. A pint of brandy given the patient over a period of a few hours shortly before admission had not caused intoxication nor alleviated the symptoms. The father reluctantly admitted that considerable quantities of white snakeroot grow on his farm, that he himself had had milk sickness in the past, and that two years previously a calf had died of trembles.

The patient was only very slightly ill; physical findings were normal, and the laboratory examinations were unremarkable. She responded promptly to parenteral fluids.

Milk Sickness in History

Milk sickness is now a curiosity accorded but a few lines in the standard medical texts. In the first half of the nineteenth century, however, it constituted a major problem on the Western frontier. Widespread interest in the disease created a voluminous literature which is catalogued in the exhaustive bibliographies of Jordan and Harris[3a], Wolf, Curtis and Kaupp[3b], and Moseley[3c]. There is a probably apocryphal belief that milk sickness was encountered by the explorer Hennepin[3a]. There is good evidence that it prevailed in North Carolina prior to 1776, and in Tennessee, Kentucky, and Ohio from the time of the earliest settlements[3b,c,4]. Half the deaths occurring in DuBois County, Indiana, in 1815, and a quarter of the deaths amongst the early settlers of Madison County, Ohio, were attributed to milk sickness[3a]. It was, of course, a scourge to cattlemen. Perhaps the most famous victim of the disease was Lincoln's mother. The relevant passage from Nicolay and Hay[5] suggests a number of corollary problems which will be subsequently discussed:

"In the autumn of 1818 the little community of Pigeon Creek [Indiana] was almost exterminated by a frightful pestilence called the milk-sickness, or, in the dialect of the country, 'the milk sick'. It is a mysterious disease which has been the theme of endless wrangling among western physicians, and the difficulty of ascertaining anything about it has been greatly increased by the local sensitiveness which forbids anyone to admit that any well-defined case has ever been seen in his neighborhood, 'although just over the creek (or in the next county) they have had it bad'. It seems to have been a malignant form of fever—attributed variously to malaria and to the eating of poisonous herbs by the cattle—attacking cattle as well as human beings, attended with violent retching and a burning sensation in the stomach, often terminating fatally on the third day. In many cases those who apparently recovered lingered for years with health seriously impaired . . . The Sparrows, husband and wife, died early in October, and Nancy Hanks Lincoln followed them after an interval of a few days."

Disastrous epidemics such as these were frequent, and if they did not kill the pioneer or force him back to his native home, they drove him still further westward—as was the case with the Lincoln family[6]. Outbreaks have been recorded at one time or another in western North Carolina, Tennessee, West Virginia, Georgia, Pennsylvania, Ohio, Indiana, Illinois, and Missouri[3c].

During the past seventy-five years the disease has virtually disappeared from older and intensively cultivated areas, for the simple but belatedly recognized reason that white snakeroot has been eradicated. Nonetheless, the disease persists endemically in certain remote and less developed agricultural regions. Milk sickness has probably occurred annually for the past one hundred and seventy years in western North Carolina, chiefly in Avery, Watauga, and Caldwell Counties, and in the adjacent areas of Tennessee[3c,7]. In White County, Tennessee, Milk Sick Mountain, which within the memory of living residents was fenced off, commemorates the pestilence[8]. During the past decade an outbreak has been reported in Illinois[9]. For the last sixty years the syndrome, known locally as alkali poisoning, has been prevalent in the Pecos Valley of New

2. McGuire, B. B.: Child Dies of Milk Sick Poisoning, Health Bull. (N. C. State Board of Health) 61:7-8 (Dec.) 1946.
3. (a) Jordan, E. O. and Harris, N. MacL.: Milksickness, J. Infect. Dis. 6:401-491 (Sept. 20) 1909; (b) Wolf, F. A., Curtis, R. S. and Kaupp, B. F.: A Monograph on Trembles or Milksickness and White Snakeroot, N. C. Agric. Exper. Station Tech. Bull. 15 (July) 1918; (c) Moseley, E. L.: Milk Sickness Caused by White Snakeroot, published jointly by the Ohio Academy of Science and the author, Bowling Green, Ohio, 1941.
4. Sackett, W. G.: The Connection of Milk Sickness with the Poisonous Qualities of White Snakeroot (Eupatorium Urticaefolium), J. Infect. Dis. 24:231-259 (March) 1919.
5. Nicolay, J. G. and Hay, John: Abraham Lincoln: A History, New York, The Century Co., 1890, v. I, pp. 30-31.
6. Stenn, F.: The Pioneer History of Milk Sickness, Ann. M. Hist. 89:23-29 (Jan.) 1937.
7. (a) Richardson, W. P.: The Milk Sickness or Trembles: Report of Five Cases, South. Med. and Surg. 93:731-732 (Oct.) 1931; (b) Milam, D. F.: Milk Sickness in North Carolina, South. Med. and Surg. 96:278-279 (June) 1934.
8. Metcalf, R. B.: Personal communication.
9. Gowen, G. H.: Milk Sickness, Illinois M. J. 74:447-452 (Nov.) 1938.

Mexico, in West Texas, and in Arizona[10].

Description of Milk Sickness

A classic description of milk sickness, which can be little improved, was published in 1810 by Drake of Cincinnati, and is reprinted by Jordan and Harris[3a]. The primary intoxication in herbivorous animals is known as "trembles." Pictures and vivid descriptions of trembles are given by numerous authorities, all of whom agree that its manifestations are identical with those of the human ailment[3a,b,10b,11]. The popular names attached to the human disease characterize it succinctly: bloody murrain, puking fever, river sickness, sick stomach, slows, staggers, stiff joint, swamp fever, tires, trembles[3a,b,4,7,9,10a,11b,12].

The latent period is unknown, but observation would indicate that the toxin exerts a cumulative effect for at least a few days. The prodromal symptoms are typically loss of appetite, languor, fatigability, and vague muscular pain. Strenuous exertion may precipitate trembling and even convulsions. This phenomenon was early noted in cattle, and before concluding a purchase the canny buyer would insist first on driving the animals hard[13]. Bowel action becomes so sluggish that obstipation is usually a prominent complaint. Persistent nausea, with a sensation of epigastric burning and heaviness, supervenes.

In the mild or chronic intoxications malnutrition may become extreme and these vague symptoms may persist for months, with ultimate spontaneous recovery. Physical overexertion or an increased consumption of contaminated milk products, however, may precipitate persistent gagging, retching, and vomiting, which soon lead to progressive dehydration, acidosis, and collapse. The individual is dull and utterly wretched, and prefers death to continued suffering. This acute phase may last from a few days to one or two weeks, and often terminates fatally. The physical findings are those of marked dehydration. The temperature is invariably normal, except perhaps terminally. There is no leukocytosis.

Convalescence is protracted by marasmus and asthenia. It is said that some individuals never fully recover their original physical capacity. Severe and sometimes fatal relapse may follow premature exertion. Characteristically, the individual becomes easily overheated, weak, and sick for months following apparent recovery.

Morbidity and mortality statistics are not available. A death rate of 20-25 per cent has been independently computed by Jordan and Harris[3a] and by Wolf, Curtis, and Kaupp[3b] in more than 300 cases culled from essentially the same sources. The mortality is, however, undoubtedly far lower if the mild and subclinical intoxications are included. On the other hand, it is likely that not a few fatal cases have been missed[12b]. Acute gastroenteritis, conversion hysteria, the chronic debilitating diseases, the intoxications, and the whole roster of causes of acidosis, coma, and convulsions must, at least from the academic standpoint, be considered in the differential diagnosis. Unfortunately there are no pathognomic physical or laboratory findings in milk sickness. The diagnosis is, however, suggested in a rural community when several members of a family who are consuming milk from the same cow or cows develop this train of symptoms.

Analysis of Cases Seen at Grace Hospital

A review of the cases of milk sickness treated at this hospital (which is located centrally in the endemic area of western North Carolina) confirms this general description. Of a total of 31 cases recorded from 1930 through 1946, 11 have been excluded because of doubtful diagnosis or inadequate records. Analysis of the remaining 20 reveals an approximately equal sex distribution. The ages of the patients ranged from 3 to 55 years, the mean being 25.

The average duration of symptoms prior to admission was two weeks. Nausea and vomiting were present in all patients, profound fatigability in a majority, constipation in about one half, and muscular pain of some description in about one third. In a number of instances there were chills,

10. (a) Marsh, C. D., Roe, G. C., and Clawson, A. B.: Rayless Goldenrod (Aplopappus Heterophyllus) as a Poisonous Plant, U. S. Dept. Agric. Bull. No. 1391 (May) 1926; (b) Marsh, C. D.: Trembles, U. S. Dept. Agric. Farmer's Bull. No. 1593 (June) 1929.
11. Clay, A. J.: Milk-Sickness, in Tice's Practice of Medicine, Hagerstown, Md., W. F. Prior Co., v. VIII:123-130, 1941.
12. (a) Walsh, W. E.: "Milk Sick," or White Snakeroot Poisoning, J.A.M.A. 87:555-556 (Aug. 21) 1926; (b) Walsh, W. E.: Milk Sickness, Illinois M. J. 58:434-437 (Dec.) 1930; (c) Wilkinson, G. R.: Milk Sickness, South. M. J. 18:797-800 (Nov.) 1925; (d) Knight, H. W.: Milk Sickness Due to White Snakeroot (Eupatorium Urticaefolium), South. Med. and Surg. 97:142-143 (March) 1935.
13. Way, J. H.: The Clinical History, Nature, and Treatment of "Milk-Sickness," Am. J. M. Sc. 96:307-316 (Sept.) 1893.

trembling, and emotional instability. Five patients became stuporous or semicomatose prior to hospitalization. The two fatalities fell in this group. In 3 cases there had been convulsions.

Physical examination in the majority of cases showed the temperature to be subnormal; in no instance was a temperature in excess of 100.2 F. recorded. The pulse and respirations were for the most part normal. The blood pressure determinations recorded were within normal limits. Varying degrees of dehydration were noted in a majority of the patients; in 6 there was obvious evidence of acidosis. In one instance a typical pleuritic friction rub was noted, doubtless of the type recently described in diabetic precoma[14]. A scaphoid abdomen and epigastric and lumbar tenderness were frequent findings.

Minimum laboratory examinations were performed in about one half the cases. Where urinalyses were recorded, the urine was consistently acid and of high specific gravity; albuminuria was present in a third of the entire series, and acetonuria in a third. In all cases the leukocyte, erythrocyte, and hemoglobin levels were essentially normal. A few nonprotein nitrogen determinations were done, and in only one instance was an elevation found. The few blood sugar determinations done were all normal.

The average duration of hospitalization was one week. Autopsy was not performed in either of the fatal cases.

Etiology

Drake correctly attributed milk sickness to the ingestion by cows of an unknown herb. His contemporaries, however, entertained other theories which to some extent remain locally current: that dew-laden grass is poisonous; that miasmas arise from polluted soil; that toxic amounts of aluminum, arsenic, cobalt, or lead are present in the grass of dangerous pastures; that cattle may be infected by noxious flies, spiders, or mushrooms[3]. It was long thought in Pecos Valley that alkaline minerals cause the disease. As early as 1840, isolated individuals had recognized that cattle who eat white snakeroot come down with trembles and that human beings who drink their milk in turn develop milk sickness[3]. An authentic instance of human intoxication caused by the accidental ingestion of white snakeroot was recorded in the mid-century[4]. Not until 1906, however, was the toxicity of white snakeroot to animals critically demonstrated by Moseley[3c]. His work was promptly refuted by Crawford, who unfortunately employed aqueous extracts of the weed[15]. His negative report, sponsored by the Department of Agriculture, delayed recognition of the etiology of the disease. Shortly afterwards, Jordan and Harris investigated an outbreak of trembles in the Pecos Valley. From animal material, feces, and soil they isolated the so-called Bacillus lactimorbi, which in two impressive reports was proposed as the pathogenic agent of milk sickness[3a,16]. The fact that milk sickness exists in the Southwest despite the absence of white snakeroot lent further plausibility to their contention. The failure of others to confirm their findings, and the strong resemblance of B. lactimorbi to the ubiquitous hay bacillus have caused the infectious theory to be relegated to the medical limbo.

Clay[11] in 1914, Marsh and Clawson[17] in 1917, Wolf, Curtis and Kaupp[3b] in 1918, and Sackett[4] in 1919 incontrovertibly established that ingestion of white snakeroot consistently produces trembles in a variety of experimental animals. Finally, in 1919, Marsh, Roe and Clawson[10a] demonstrated that the rayless goldenrod of the Southwest likewise causes trembles in the experimental animal. A sibling of this plant, Aplopappus fructicosus, has been subsequently found to be equally toxic[18].

The proposition that milk sickness is transmitted to man by the milk of cows which have ingested either white snakeroot or rayless goldenrod has never been experimentally established, for obvious reasons. The evidence is circumstantial, but nonetheless overwhelming. Gowen states that in each of 21 cases of milk sickness investigated

14. Armanino, L. P. and Ory, E. M.: Acute Pleurisy as a Dehydration Phenomenon in Diabetic Precoma, Am. J. M. Sc. 211:597-601 (May) 1946.

15. Crawford, A. C.: The Supposed Relationship of White Snakeroot to Milk Sickness or "Trembles," U. S. Dept. Agric. Bureau of Plant Industry, Bull. No. 121:3-20 (April 18) 1908.

16. (a) Jordan, E. O. and Harris, N. MacL.: The Cause of Milk Sickness or Trembles, J.A.M.A. 50:1665-1673 (May 23) 1908; (b) Luckhardt, A. B.: Additional Notes on the Bacteriology and Pathology of Milksickness, J. Infect. Dis. 6:492-505 (Sept. 20) 1909.

17. Marsh, C. D. and Clawson, A. B.: Eupatorium Urticaefolium as a Poisonous Plant, J. Agric. Research 11:699-716 (Dec. 24) 1917.

18. Couch, J. F.: Trembles (or Milk Sickness), U. S. Dept. Agric., Circ. No. 306 (Nov.) 1933.

by him, cattle had been turned to forage in wooded areas where white snakeroot was identified; the animals developed trembles, and in turn those who drank their milk developed the intoxication[9]. Culpepper describes his own case of milk sickness, acquired from the milk of his cow, which three days previously had eaten rayless goldenrod[19]. Numerous observers have claimed that the meat of animals dead of trembles is toxic, although at least two investigators have failed to substantiate this belief[3b,4].

Tremetol

In 1927 Couch announced the successful extraction from white snakeroot of a pure toxic substance, which he called tremetol[20]. This substance uniformly causes trembles in the experimental animal. In 1930 he isolated tremetol from the rayless goldenrod[21]. Tremetol is obtained, by a series of alcohol and ether extractions, as an aromatic, straw-yellow, thick oil. The compound is a levorotatory secondary alcohol. The probable empirical formula is $C_{16}H_{22}O_3$ and includes a phenyl ring with a sidechain containing two double bonds. According to Couch, it can be recovered in quantity from both dry and fresh rayless goldenrod, but only from fresh white snakeroot, although it has been long maintained that the dry weed remains poisonous. Although readily oxidized, tremetol is unaffected by the heat of pasteurization. Two other toxic substances are contained in snakeroot, but tremetol alone produces trembles.

Tremetol has not been identified in contaminated milk, although Couch has produced trembles in experimental animals with butter made of contaminated milk[22]. It is thought that in the course of metabolism some change is wrought in its configuration without alteration of toxicity. Presumably lactation exerts a protective effect in the cow, for cows which have given poisonous milk for some time may not be affected by trembles until the milk supply fails. It has frequently been observed that the severest cases of milk sickness are occasioned by an inordinate consumption of cream and butter, and it is probable that tremetol occurs in high concentration in the fatty part of the milk. The local mountaineers believe that the toxin may be seen as minute greenish globules on the top of milk.

19. Culpepper, M. B.: Milk-Sickness Erroneously Called Alka'i Poisoning, Southwestern Med. 10:391-393 (Sept.) 1926.
20. Couch, J. F.: (a) The Toxic Constituent of Richweed or White Snakeroot (Eupatorium Urticaefolium), J. Agric. Research 35:547-576 (Sept. 15) 1927; (b) Milk Sickness. The Result of Richweed Poisoning, J.A.M.A. 91:?34-236 (July 28) 1928; (c) Tremetol, The Compound that Produces "Trembles" (Milksickness), J. Am. Chem. Soc. 51: 3617-3619 (Dec.) 1929.
21. Couch, J. F.: The Toxic Constituent of Rayless Goldenrod, J. Agric. Research 40:649-658 (April 1) 1930.
22. Couch, J. F.: Trembles (Milk Sickness) Produced by Toxic Butter, Vet. Med. 36:244-245 (May) 1941.

Description of Plants Responsible for Milk Sickness

In the light of present knowledge, it is evident that trembles and hence milk sickness may occur in any area where white snakeroot or rayless goldenrod abounds. Of the forty-odd members of the Eupatorium family, white snakeroot alone is toxic. Apparently it grows only in this country, for milk sickness has never been described elsewhere. It is known popularly as white or Indian samicle, richweed, deerwort, boneset, white top, squaw weed, steria, and poolwort[3c,23]. Its season is from July through November. It grows in rich, moist soil, such as is found in timbered and uncleared areas. Locally, at least, it luxuriates in the wet bottoms of mountain coves. Cattle dislike it but will eat it of necessity in unusually dry spells, when they are driven to forage in shaded, still verdant areas. Hence milk sickness most commonly occurs in dry years. The weeds in dry hay are said to remain poisonous[1]. The plant grows throughout the eastern half of the United States. The variation in toxicity from year to year and from region to region has not been satisfactorily explained[20b].

Cultivation of the soil eliminates the weed. Although the region served by this hospital has been farmed for many generations, many of the upland pastures remain only partially cleared, and here the plant may often be found. Few farmers will admit the presence

23. "It blooms in September, and has a white blossom of the corymb type, consisting of a cluster of flowers, each on its own foot stalk and arising from a common axis, and each having a fringy bloom. The stalk is from one to four feet high, with a square stem, and has opposite petioled leaves broadly oval at the base, taper-pointed, coarsely toothed, three-ribbed and veined, thin and easily skeletonized by insects . . . The leaves grow in pairs from alternate sides of a stalk, so that each pair is at right angles to the pair above or below it . . . The roots . . . grow only about three inches deep . . . The plant thrives well in dry weather, the leaves and stem remaining green when neighboring plants are parched."[11]

of snakeroot on their property—often because they do not recognize the plant or refuse to believe that it causes milk sickness, but more often because the very rumor vastly depreciates the resale value of their land.

Rayless goldenrod or jimmy weed is found from southern Colorado to the Texas Panhandle and south to Arizona, Sonora, and Chihuahua[24]. It grows luxuriantly along irrigation ditches, and, like white snakeroot, is eaten by cattle only in lieu of more palatable fodder[10a].

Pathology and Pathologic Physiology

Postmortem examinations have been reported on only 3 human victims of milk sickness[3a]. The few detailed protocols are on animals. The salient findings have been consistently enlargement and fatty metamorphosis of the liver, occasionally so extreme as to render the histologic structure almost unrecognizable; fatty infiltration of the heart; acute parenchymatous nephritis; hyperemia of the stomach and intestines, and hyperplasia of Peyer's patches[3a,b]. The human subjects showed essentially the same changes. Adequate studies of the brain have not been made.

The morbid physiology of milk sickness is obscure, since the disease is now confined to remote rural areas where laboratory facilities are inadequate. Walsh, early in this century, was apparently first to recognize the acidosis[25]. He has since suggested that the toxin impairs the power of the liver to store glycogen, and that hypoglycemia is of constant occurrence. The evidence to support this hypothesis is that one of his convalescent patients was found to have a normal glucose tolerance test but an impaired levulose tolerance test, and that 3 patients with acute milk sickness were found to have rather low blood sugars, despite previous treatment with glucose[12b].

The degree of acetonuria produced in the experimental animal by the ingestion of fresh white snakeroot is said by Bulger, Smith, and Steinmeyer to be far greater than that produced by starvation alone. These workers found hyperlipemia and hypoglycemia, with terminal convulsions, to be frequent in the rabbit, although these effects were by no means regularly produced. Their animals exhibited no impairment of nitrogen metabolism[26]. Cartland, Heyl, and Neupert, using dried white snakeroot, were unable to reproduce the severe hypoglycemia described by Bulger and his coworkers, and found only a mild, irregular hypoglycemia without the production of trembles or any toxic effect on the liver and kidneys[27]. Couch, working with poisoned sheep, has found a persistent hyperglycemia[20a]. Pritchett, of this hospital, has likewise found hyperglycemia in the fatally poisoned sheep[28].

The accumulation of guanidine as a result of tissue destruction has been proposed as an explanation of the action of snakeroot[29]. The possibility that tremetol may also be a neurotoxin has not been explored. No plausible explanation of the pathogenesis of the acute intoxication or of the prolonged, if not permanent, disability following apparent recovery has yet been suggested. A fascinating, if somewhat recondite, avenue of further investigation lies open.

Treatment

Treatment at present remains entirely empirical. The traditional lay treatment in these mountains has long been the administration of honey and brandy to the point of extreme intoxication—a practice in Hardin's opinion not lightly to be dismissed. In his experience cases not so treated have a poorer prognosis[1]. Clay[11], Lambert[30], and Way[13], to name a few, likewise warmly ad-

24. "The plant is a stout, erect, tufted perennial herb with a shrubby or near shrubby base, minutely hairy to nearly smooth, commonly from one to two feet in height, but sometimes under favorable circumstances of growth, as on banks of irrigating ditches, it may reach a height of four feet or even more.

"The leaves are alternate, linelike or broader, one-eighth to one-fourth inch wide, three-fourths to two and one-half inches long, usually with stout, stiff hairs on the margin, occasionally with short, sharp, scattered teeth, and acute or obtuse at the apex.

"The heads are numerous, with or without stems, in terminal flat-topped bunches; the bracts surrounding the flower heads are about one-eighth inch high, oblong, usually obtuse, with obscurely green tips. The tips have seven to fifteen tubular yellow flowers. The seeds are silky and small."[10a]

25. Editorial: Milk Sickness, or "Trembles," J.A.M.A. 87: 1835 (Nov. 27) 1926.

26. Bulger, H. A., Smith, F. M. and Steinmeyer, A.: Milk Sickness and the Metabolic Disturbances in White Snakeroot Poisoning, J.A.M.A. 91:1964-1966 (Dec. 22) 1928.

27. Cartland, G. F., Heyl, F. W. and Neupert, E. F.: The Hypoglycemic Properties of White Snakeroot (Eupatorium Urticaefolium), J. Am. Pharm. A. 20:448-454 (May) 1931.

28. Pritchett, J. B.: Unpublished data.

29. Cutter, J. T., cited by Moseley[3c].

30. Lambert, C. F.: Milk Sickness, South. Med. and Surg. 99: 165-166 (April) 1937.

vocate this treatment, although other workers have obtained good results by the enteral and parenteral administration of water, physiologic saline solution, crude or pure glucose, and sodium bicarbonate. The rationale of the brandy treatment is that tremetol is partially detoxified by esterification with the alcohol[1]. Certainly nausea is alleviated by the induction of an alcoholic stupor. It remains to be proved, however, that vomiting is a local rather than a central effect of tremetol, or that a significant amount of unfixed toxin is present in the stomach by the time treatment is instituted. It would appear reasonable that similar improvement might be obtained by heavy barbiturate narcosis. Evacuation of the bowels is generally recommended.

The therapeutic regimen employed by Hardin at this hospital consisted in the administration of brandy and honey to the point of intoxication, duodenal drainage, proctoclyses of sodium chloride and sodium bicarbonate, hypodermoclyses of glucose solution, enemas, and magnesium sulfate catharsis. After alcoholic intoxication was maintained for twenty-four to seventy-two hours, the patient was usually able to tolerate a liquid and later a soft diet without vomiting, and to leave the hospital within a week. Since Hardin's death in 1937, treatment has consisted solely of honey and brandy given if necessary by gavage, intravenous infusions, and emptying of the bowel as it is deemed necessary. Equally good results have been attained.

Prevention is, of course, the ultimate goal of all treatment, and the history of milk sickness indicates that elimination of the poisonous plants has led to the virtual disappearance of the disease. It persists in remote rural areas because of prejudice, ignorance, and indifference. Since these phenomena are not regional, it may be safely assumed that milk sickness will continue to occur sporadically. Fortunately the disease has not been known to occur in urban communities (where it would probably be undiagnosable), since commercial pooling of milk renders the toxin innocuous by dilution.

Summary

Milk sickness is now a medical curiosity, although a century ago it was a formidable disease. The primary intoxication in cattle is caused by the ingestion of white snakeroot or rayless goldenrod, and is transmitted in milk to man. The disease is characterized by anorexia, weakness, fatigability, constipation, nausea, and pernicious vomiting in acute, relapsing, and probably chronic forms. The acute phase is marked by profound dehydration and acidosis, and a fatal termination is common. Fever and leukocytosis do not occur.

The toxic principle contained in white snakeroot and rayless goldenrod is a secondary alcohol called tremetol. The mode of action of tremetol is unknown. Treatment is nonspecific and is directed toward correction of dehydration and acidosis. Empirically, the use of intoxicating doses of alcohol apparently improves the prognosis. Convalescence is prolonged, and relapse often follows premature physical exertion.

Milk sickness has virtually disappeared as white snakeroot has been eliminated from the pasture land of the eastern United States.

DIPHTHERITIC MYOCARDITIS

A Case Report

WESTON M. KELSEY, M.D.
WINSTON-SALEM

Myocarditis is a serious complication of diphtheria. Because of the decreasing incidence of diphtheria in the past few years, patients with diphtheritic myocarditis are seen only rarely. The case reported below illustrates many of the problems which arise in the diagnosis and treatment of this disease.

Report of Case

E. H., a 13-year-old boy, was referred to the North Carolina Baptist Hospital on December 21, 1945, with the diagnosis of pharyngeal diphtheria. The history obtained was unsatisfactory, since he lived with a foster mother who knew little about him. As almost all North Carolina school children are immunized against diphtheria, he probably had received prophylaxis. His present illness began about six days prior to admission, with general malaise. Five days before admission the right side of his neck became "enormous." Two days later a right peritonsillar abscess was incised at the local hospital. He remained in that hospital for three days, during which time he received 240,000 units of penicillin. When a virulent diphtheria organism was obtained from the throat, he was given 25,000 units of diph-

From the Department of Pediatrics of the Bowman Gray School of Medicine of Wake Forest College, and the Pediatric Service of the North Carolina Baptist Hospital, Winston-Salem, North Carolina.

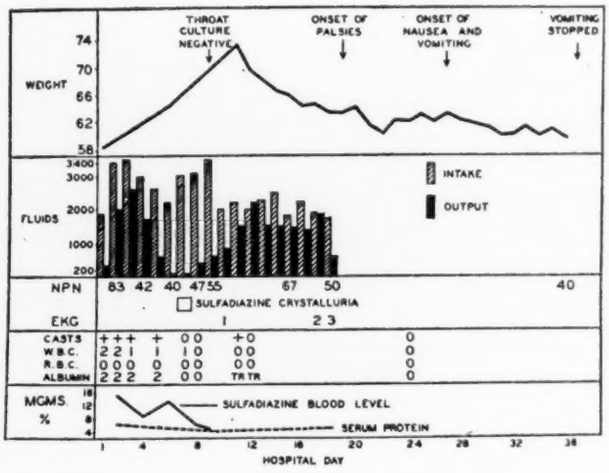

Fig. 1

theria antitoxin and was tranferred to this hospital for further care.

Physical examination on admission revealed a poorly nourished, moderately ill, white male in no acute distress. The rectal temperature was 99.2 F., the pulse rate 88, and the respiratory rate 20. The blood pressure was 110 systolic, 60 diastolic. The patient was dehydrated. The cervical glands were greatly enlarged and slightly tender. A necrotic membrane covered the right tonsil and extended to the uvula; a smaller membrane was present on the left tonsil, and the entire pharynx was edematous. The palate moved poorly, and the patient was hoarse; this hoarseness was felt to be a result of local edema. The heart was not enlarged to percussion; the rate was regular and no murmurs were heard. The liver and spleen were not palpable. The neurologic examination was within normal limits.

Laboratory examinations made on admission showed the hemoglobin to be 13.7 Gm., the red blood cell count 5,040,000, and the white blood cell count 13,800, with 84 per cent polymorphonuclear leukocytes. The urinalysis showed a 2 plus reaction for albumin; the specific gravity was 1.024. The centrifuged sediment contained a moderate number of granular casts, many white blood cells, and rare red blood cells. Throat cultures showed a moderate number of beta hemolytic streptococci, and numerous colonies of Corynebacterium diphtheriae, which gave a positive guinea pig virulence test. No attempt was made to establish the type of the organism. The remainder of the laboratory data are presented on the graph (fig. 1).

No further antitoxin was given, since it was thought that the patient had received an adequate amount prior to admission. Sulfadiazine and penicillin were given for the streptococcal infection. Large amounts of parenteral fluids were administered to combat the dehydration.

The initial azotemia, which was felt to be caused by dehydration, was relieved by the administration of fluids. A second episode of azotemia following renal complications of sulfadiazine therapy was treated successfully by stopping the sulfadiazine and by giving increased amounts of fluids and alkalies.

Cardiac involvement was not suspected until the sixth day after admission. Physical examination on that day showed no clinical signs of cardiac insufficiency. The pulse rate was 100 and regular. The heart was not enlarged to percussion; the sounds were normal, and there were no murmurs. The blood pressure was 100 systolic, 60 diastolic. An electrocardiogram was obtained because it was felt that early cardiac failure might be a factor in the patient's oliguria. The first electrocardiogram (fig. 2) showed low voltage in the limb leads, a PR interval

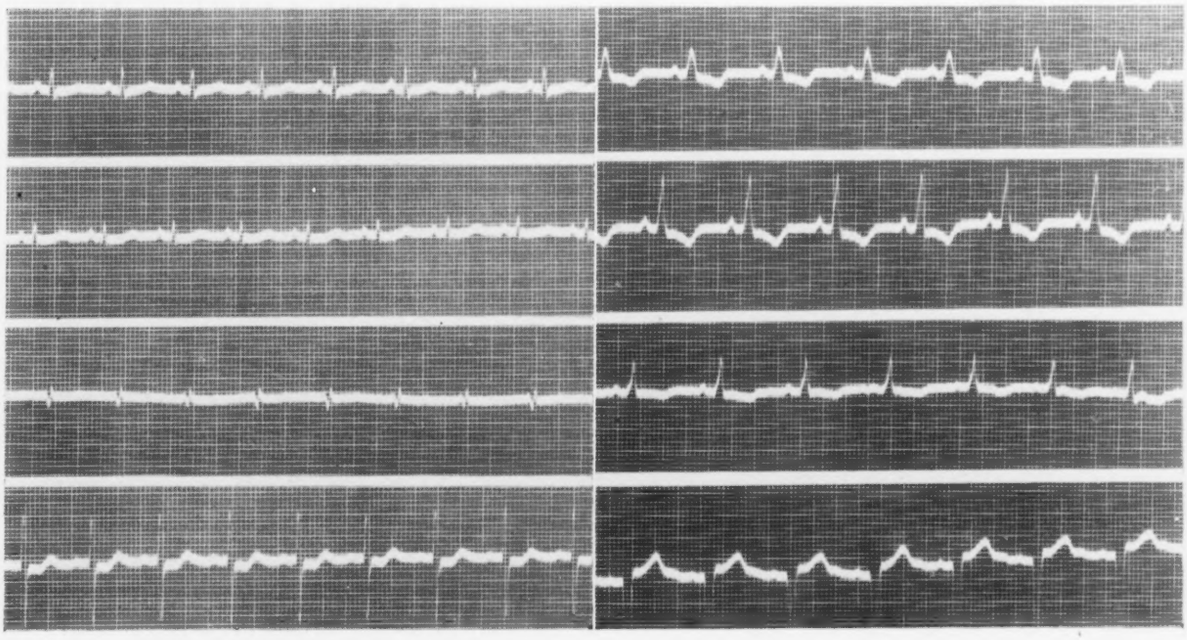

Fig. 2. December 31, 1945. Normal sino-auricular rhythm. Rate 100. Low voltage in limb leads. PR interval 0.16 seconds. QRS time 0.05 seconds. T waves in all leads small but upright. Lead C F IV normal save for 1 mm. depression of S-T segment.

Fig. 3. January 8, 1946. Comparison with figure 2 shows extreme changes. Rate 80. Delayed interventricular conduction is evident (QRS time 0.10 seconds), and the T waves in the limb leads are inverted. S-T_4 is elevated 1.5 mm., and there is no positive R_4 deflection. The PR interval is unchanged—0.16 seconds. The patterns are not unlike atypical bundle-branch block and are consistent with an acute process in the myocardium.

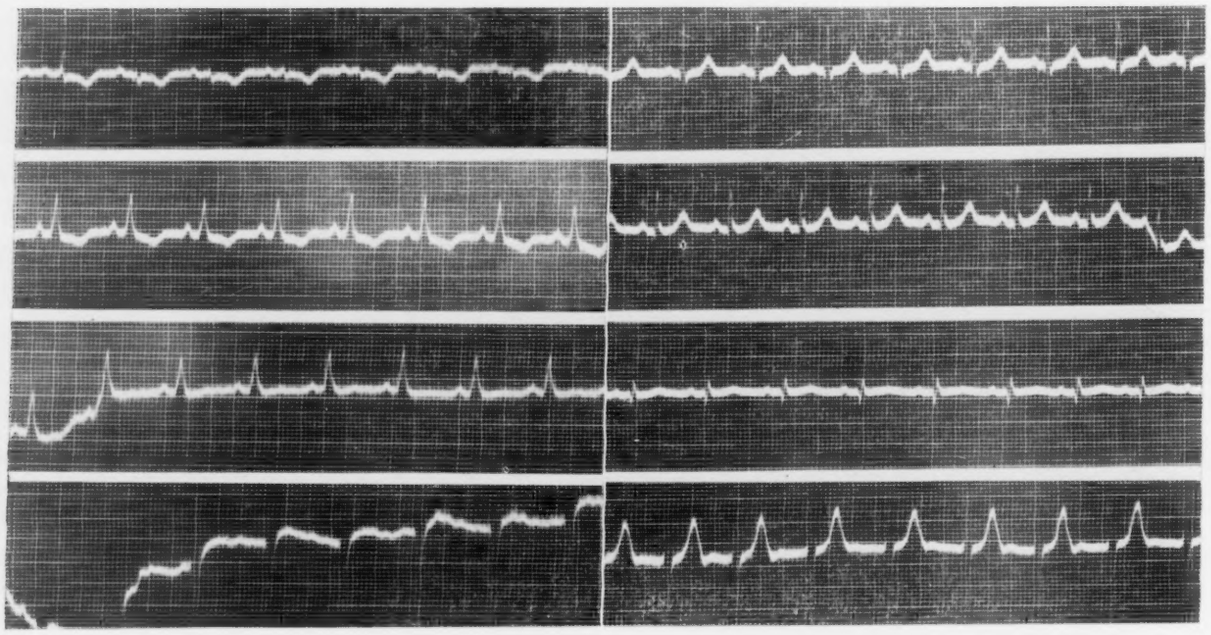

Fig. 4. January 10, 1946. This electrocardiogram, made forty-eight hours after fig. 3, shows further drastic changes. QRS_1 shows markedly lowered voltage; $S\text{-}T_1$ is depressed and there is cove-plane inversion of T_1. T_3 is now upright and T_4 is somewhat flattened. Positive R_4 deflection is still absent. These findings indicate a rapidly changing acute process in the myocardium.

Fig. 5. March 10, 1946. Ten weeks later the electrocardiogram is normal. The voltage is adequate; the T waves are upright and of good caliber; QRS time is 0.07 seconds, and the PR interval is unaltered—0.16 seconds.

at the upper limits of normal, and a slight depression of the S-T segment in C F IV. The second electrocardiogram (fig. 3) showed a marked increase in the QRS time to 0.10 seconds, inversion of the T waves in the limb leads, elevation of $S\text{-}T_4$ to 1.5 mm., and absence of a positive R_4 deflection. Two days later there was marked lowering of the voltage in QRS_1, together with depression of $S\text{-}T_1$, coveplane inversion of T_1 and absence of a positive R_4 deflection. These changes disappeared gradually, and ten weeks later the electrocardiogram was entirely normal (fig. 5).

Questionably weak heart sounds were heard occasionally and moderate tachycardia was noted, but physical examination never revealed definite evidence of heart disease. Nausea and vomiting were troublesome from the twenty-seventh to the thirtieth day, and it was felt that this was a symptom of myocardial disease[1]. During this patient's prolonged course no cardiac enlargement was demonstrated either by percussion or by teleroentgenogram. The blood pressure remained normal. The liver never became palpable or tender, and the cardiac rhythm and rate remained regular.

On admission it was felt that the local edema caused the hoarseness and the weak palatal movement. Improvement of these functions in the first few days of hospitalization substantiated this opinion. Twenty-two days after the onset of the illness, however, the patient again became hoarse. The gag reflex was not present, and fluids were regurgitated through his nose. This palatal paralysis persisted until the fifty-first day, from which time recovery was rapid.

The patient was seen seven weeks after discharge. During this interval he had gained 10 pounds in weight, and there was no edema. Physical examination revealed a normal 13-year-old boy. The Schick test was negative, and an electrocardiogram was normal. The patient was discharged to full activity.

1. McMillan, R. L., Cowden, F. E., and Reinhart, J. B.: Gastrointestinal Symptoms of Progressive Myocardial Disease. Am. Heart J. 29:580-587 (May) 1945.

The Frequency of Subclinical Diphtheritic Myocarditis

The development of electrocardiographic evidence of myocardial involvement in diphtheria without physical signs of cardiac disease apparently is common. The fact that electrocardiograms are usually made only after the clinical findings point to cardiac trouble probably accounts for the relatively low percentage of cases of diphtheria in which myocarditis is diagnosed, and also explains the marked discrepancies in the incidences of cardiac involvement reported in various series. Many authors who have made routine electrocardiograms on all their patients with diphtheria have found that a significant number of patients showed electrocardiographic abnormalities without physical signs of myocardial disease[2], and a number of authors comment that there is probably more undiagnosed diphtheritic heart disease than that which is recognized.

Prognosis

There is agreement that the prognosis is poorest in the presence of conduction defects.

Andersen, however, noted that the patients who died earliest had the most marked physical signs and the least electrocardiographic changes[2b]. Rostoski[3] believes that the early onset of myocarditis, even if it is manifested only by the electrocardiogram, implies a poor prognosis. Alstead, on the other hand, feels that the course is usually mild when electrocardiographic changes are present in the absence of physical findings[2a]. Certainly there is abundant evidence that routine electrocardiograms should be made early in the course of diphtheria as a guide to the management of convalescence. Intravenous hypertonic glucose is probably the most effective form of therapy in diphtheritic heart disease, and prompt institution of treatment is probably of importance.

The prognostic significance of albuminuria is doubtful. Kuenzel[4] observed that the persistence of albuminuria beyond the eighth day was of serious import. Hoyne[5] pointed out the frequency with which albuminuria precedes the onset of clinical myocarditis, and considered it an unfavorable prognostic sign. Undoubtedly dehydration, with the accompanying renal dysfunction, may cause albuminuria.

Burkhardt and his coworkers[2h], Kuenzel[4], Place[2g], and Alstead[2a] commented on the high degree of correlation between the development of myocarditis and the appearance of paralysis. Togasaki and his associates, however, observed 278 patients with diphtheritic myocarditis, of whom only 8 had paralysis[6].

Treatment

The treatment of diphtheritic myocarditis is unsatisfactory. Antitoxin should be given as early as possible, and in adequate amounts. There is agreement that intravenous hypertonic glucose can be of value in the treatment of diphtheritic myocarditis. This belief is substantiated by animal experiments[7]. It is generally agreed that digitalis is a dangerous drug in diphtheria[2f,8]. The extensive studies of Edmunds[7] on animals give additional confirmation of its potential toxicity in this disease.

Summary

A case of diphtheritic myocarditis is reported, and the frequency of subclinical diphtheritic myocarditis is discussed. The importance of early recognition of myocardial involvement is stressed, since the prognosis may be altered by appropriate therapy in the acute phase and by prolonged convalescent care.

Dr. Robert L. McMillan of the Department of Medicine interpreted the electrocardiograms and gave advice in the management of the cardiac complications.

2. (a) Alstead, S.: The Electrocardiogram in Diphtheria, Quart. J. Med. 1:277-288 (April) 1932; (b) Andersen, M. S.: Electrocardiographic Studies on Diphtheritic Myocarditis, Acta med. Scandinav. 84:253-312, 1934; (c) Natin, I. and da Rin, C.: Electrocardiographic Alterations in Diphtheria, Semana Med. 1:1789-1861 (June 14) 1934; (d) Shookhoff, C., and Taran, L. M.: Electrocardiographic Studies in Infectious Diseases: Diphtheria, Am. J. Dis. Child. 42:811-836 (Oct.) 1931; (e) Neubauer, C.: Diphtheric Heart-Disorders in Children, Brit. M. J. 2:91-94 (July 5) 1942; (e) Wesselhoeft, C.: Cardiovascular Disease in Diphtheria, New England J. Med. 223:57-66 (July 11) 1940; (g) Place, E. H.: The Heart in Diphtheria and Scarlet Fever, New England J. Med. 207:864-874 (Nov. 17) 1932; (h) Burkhardt, E. A., Eggleston, C., and Smith, L. W.: Electrocardiographic Changes and Peripheral Nerve Palsies in Toxic Diphtheria, Am. J. M. Sc. 195:301-313 (March) 1938.

3. Rostoski, O.: Heart in Diphtheria, Muenchen. med. Wchnschr. 85:434-440 (March 25) 1938.

4. Kuenzel, O.: Value of Albuminuria in Prognosis of Diphtheria, Deutsche med. Wchnschr. 65:328-330 (March 3) 1939.

5. Hoyne, A. L., and Welford, N. T.: Diphtheritic Myocarditis, J. Pediat. 5:642-653 (Nov.) 1934.

6. Togasaki, Y., Rosove, L., Bower, A. G., and Hamilton, P. M.: Treatment, Complications and Deaths in 753 Cases of Clinical Diphtheria, Am. J. M. Sc. 204:218-227 (Aug.) 1942.

7. Edmunds, C. W.: Circulatory Collapse in Diphtheria, Am. J. Dis. Child. 54:1066-1079 (Nov.) 1937.

8. McCulloch, H.: Administration of Digitalis into Children with Diphtheria, South. M. J. 14:110 (Feb.) 1921.

Maternal Welfare Section

MATERNAL MORTALITY IN NORTH CAROLINA

FRANK R. LOCK, M.D.

WINSTON-SALEM

Obstetrics is an inevitable part of the practice of most physicians, and each general practitioner conducts about 50 deliveries a year, or approximately 2,000 during his medical experience. By the law of averages he sees the frequent complications of pregnancy often, but may never encounter other less common complications. The Maternal Welfare Committee has collected data from all the physicians in North Carolina who have managed an obstetric condition resulting in a maternal death. The collected information may benefit all who are interested in obstetrics, for by learning the facts associated with

Report of the Maternal Welfare Committee to the Medical Society of the State of North Carolina, read at the Second General Session, Virginia Beach, May 14, 1947. Members of the Maternal Welfare Committee are:

J. Street Brewer, M.D.
G. M. Cooper, M.D.
F. W. Franklin, M.D.
J. S. Hunt, M.D.
T. L. Lee, M.D.
Ivan Procter, M.D.
R. A. Ross, M.D.
R. A. White, M.D.
Frank R. Lock, M.D., Chairman

each death we may make an intelligent approach to correct the specific problems which now exist.

In December, 1945, the Maternal Welfare Committee was organized. At that time North Carolina was among the eight states in the nation with the highest maternal death rates. The object of the committee is to promote any method which will reduce the number of deaths associated with pregnancy.

One hundred and twenty-seven deaths have now been studied by the committee. A questionnaire has been sent to each physician who has signed a death certificate for a woman whose final illness was associated with pregnancy. Often additional questions are asked to obtain conclusive evidence of the cause of the patient's death and the method of management, since the committee must have full information in order to analyze the record accurately. A meeting of the committee is held whenever a sufficient number of complete records are on hand. The entire committee considers carefully each detail of the patient's last illness and of the treatment the patient received. No names are used in the presentation and discussion of the record, and the entire committee decides in an unprejudiced manner whether or not the death was preventable, and the factor responsible for those deaths which are preventable. The final report to the physician who signed the death certificate is outlined, and in each instance a report giving the conclusions of the committee and their reasons for them is sent to the physician.

Principal Causes of Maternal Deaths

Toxemia

Toxemias—hypertensive complications of pregnancy—have been the direct cause of death or a contributory factor in an astounding majority of the cases (table 1). In 93 instances the committee has had to consider this important problem as a factor in the patient's last illness and in the decision made in the management of the presenting problem.

Table 1

Toxemia of Pregnancy

Contributing to death	41
Direct cause of death	52
Convulsions: Before delivery	16
After delivery	14
Nephritis	22
TOTAL	93

Twenty-two of the patients clearly demonstrated renal insufficiency complicated by pregnancy, with a definite record of hypertension and albuminuria in the early weeks of pregnancy. In some instances, they were properly advised to have the pregnancy interrupted, but refused. In others, the lack of adequate facilities for the care of the patient or an error in judgment, with inadequate appreciation of the danger to the patient, resulted directly in the loss of life.

Acute and fulminating pre-eclampsia followed by convulsions occurs in an appreciable group of patients in our state. Many physicians consider one examination during each month of pregnancy sufficient to fulfill the obligation of prenatal care. The fallacy of this concept is clearly demonstrated by the convulsive deaths of five women within two weeks after an examination at which no abnormality was found. In order to avoid these deaths, urinalyses and determinations of the weight and blood pressure should be made each week during the last month of pregnancy, and the patient should be educated to the danger related to signs and symptoms of toxemia. The observance and treatment of minor changes will almost always forestall the development of a serious toxemic condition.

Thirty patients had convulsions during their last illness. These preceded delivery in 16 cases, and in 14 followed a successful delivery. Not one of this group had the advantage of ideal prenatal care, and in only one instance was the death considered by the committee to be unpreventable. In some cases, however, the death was unavoidable with the available facilities and assistance. The principal error noted in the management of this problem was a false sense of security after delivery of the patient and inadequate treatment in the early puerperium.

On five occasions sudden death has followed the administration of a spinal anesthetic to a patient with toxemia. The committee is convinced that this anesthetic method is contraindicated in the presence of hypertension and pregnancy.

Hemorrhage

Hemorrhage closely follows the toxemias of pregnancy as a cause of maternal deaths. In 75 cases it has been the direct or contributing cause of death (table 2). Postpartum

Table 2

Hemorrhage

Contributing to death	24
Direct cause of death	51
Postpartum hemorrhage	31
Postabortion hemorrhage	9
Ectopic pregnancy	5
Placenta praevia	5
Abruptio placentae	1
TOTAL	75

hemorrhage has taken the greatest toll of lives. Thirty-one women have bled to death following their delivery, 9 after an abortion, and 5 as a result of ectopic pregnancy. In 24 patients the loss of blood has contributed seriously to the fatal outcome of an infection or other complication. Only 6 of these patients have received blood transfusions, and none have been given a quantity of blood sufficient to be expected to save the patient's life. Seven of the patients have died unattended by a physician. Delays in calling for assistance on the part of the family were responsible in 3 cases, but in 4, a precipitate labor was quickly followed by a profuse hemorrhage and death. The latter patients in rural areas were the victims of their environment and could have been saved only by proper assistance immediately available.

Abortions

Abortions represent a widespread menace in our state. Criminal interference with pregnancy cannot be proven in most instances, but it is strongly suggested in many records. Of the 24 deaths associated with abortion, 9 were due to hemorrhage, 11 to infection, and 4 to toxic drugs ingested in an attempt to produce an abortion (table 3).

Table 3

Causes of Deaths Associated with Abortions

Infection	11
Hemorrhage	9
Poison	4
TOTAL	24

Nine (45 per cent) of the patients were married, leaving other children motherless. Often the patient concealed her illness at home, and medical aid was not sought until she was moribund. This fact is shown by the predominance of abortions in the deaths from infection. The majority of the patients in the group received little or no treatment until the last forty-eight hours of life.

Heart disease and anesthesia

The two additional complications requiring our attention within the state are heart disease and anesthetic complications, which together took 21 lives. The records show that the cardiac lesion was not often recognized early, and that proper treatment was delayed or withheld through errors in diagnosis or management. Each of the commonly used anesthetic methods has been responsible for one or more deaths. Spinal anesthesia has been the principal offending agent, with sudden death following the injection on seven occasions.

Factors Responsible for Preventable Deaths

As a society we must be vitally interested in the factors responsible for our preventable maternal deaths (table 4). Eighty-two records have been fully classified by the committee. Sixty-six of the group were obstetric deaths, or deaths resulting directly from a complication of pregnancy. Sixty of these were preventable. Sixteen were classified as non-obstetric, since the cause was not directly related to pregnancy, although the death occurred because of the presence of pregnancy. Five of these were classified as preventable.

Table 4

Preventable obstetric deaths	60
Non-preventable obstetric deaths	6
Preventable non-obstetric deaths	11
Non-preventable non-obstetric deaths	5

One or more of the three principal factors listed in table 5 was present in each of the preventable deaths. Ignorance or neglect on the part of the patient or her family (not seeking medical care or refusal to follow the correct advice when it was given) was the most frequent factor in the records. In 55 cases this problem was largely responsible for the patient's death. Errors in judgment or technique on the part of the physician were recorded on 48 cases by the committee. Lack of facilities to care for the patient has directly caused 11 deaths.

It is recognized that we have observed idealistic rules in our assignment of the responsible factor. Under ideal conditions peritonitis does not develop following a

Table 5

Factors Responsible for Preventable Deaths

Neglect or ignorance of patient	55
Physician error	48
Lack of facilities	11

cesarean section or vaginal delivery. The advent of this complication is an indication of a break in aseptic technique; and although an error in sterilization of the instruments is at fault, it is the responsibility of the physician in charge of the patient. In other instances gross errors in management have contributed to the patient's death.

The need for more capable consultants is a paramount problem. In many areas it is impossible to obtain the consultation of a specialist, and often telephone or "curbstone" consultations have been used. In only 12 of the 72 records which gave full information relative to consultations was satisfactory consultation obtained during the patient's terminal illness (table 6). This problem and a lack of interest in exact assignment of the cause of death are emphasized by the filing of 22 death certificates on which the cause of the patient's death was given inaccurately.

Table 6

Consultation

Adequate consultation	12
Unsatisfactory or none	60

Summary and Conclusions

It is too early in the course of the work of the Maternal Welfare Committee to draw sweeping conclusions about the general problems which exist in North Carolina. Several limited conclusions are obvious, however.

1. Widespread ignorance exists among the laity. A broad educational campaign must be launched to avoid unnecessary deaths which result from the patient's failure to seek medical aid.

2. Standard practices in the management of obstetric complications must be widely encouraged by all members of our profession, and the rules of conservatism must be closely adhered to.

3. Available consultants are urgently needed in many areas.

4. Blood banks or other measures to facilitate the administration of whole blood are essential to safe obstetric practice and must be made available.

5. A careful general examination of each patient, including an examination of the heart and lungs, must be made to avoid unnecessary deaths after anesthesia and minor operative procedures incidental to obstetric problems.

6. Ten deaths directly due to anesthesia re-emphasize the danger of all anesthetic agents. Careful preparation of the patient and skillful administration, with all safeguards necessary to protect the patient, are required to make anesthetization a safe procedure.

Prevention versus cure.—For a very long time we have accepted the old adage, "Prevention is better than cure." In our new era the belief in it—for of its truth there can be no doubt—must be made ever more manifest in our research and its directives and in our teaching. The most conspicuous interest of the student ten or twenty years hence will, I hope, no longer be in the rare or difficult and too often incurable case, but in the common and more understandable and preventable disease. May the daily question on his lips become not "What is the treatment?", but "What are the causes?" and "If preventable, then why not prevented?"—John A. Ryle: Social Pathology and the New Era in Medicine, Bull. New York Acad. Med. 23:328 (June) 1947.

THUMBNAIL SKETCHES OF EMINENT PHYSICIANS

JOSIAH C. TRENT, M.D., F.A.C.S., *Editor*

OBSTETRICS AND GYNECOLOGY IN AMERICA

VII

CHARLES D. MEIGS AND HUGH LENOX HODGE

Medical history records that some men are remembered for their obstruction to the progress of medicine. Charles D. Meigs (1792-1869), an inspired teacher of obstetrics and gynecology in the Jefferson Medical College, Philadelphia, is a typical example. He stubbornly and intolerantly opposed the introduction of anesthesia into surgery and obstetric practice, the theory of contagion of puerperal infection, cesarean section, and celiotomy for ovarian and other pelvic neoplasms. Time has proven that these measures were momentous and enduring in significance.

In 1843, Oliver Wendell Holmes presented his essay on the "Contagiousness of Puerperal Fever" before the Boston Society of Medical Improvement. Immediately Professor Meigs and Professor Hugh L. Hodge of the University of Pennsylvania attacked Holmes's theory. Meigs, a fine orator, berated this "absurd theory" and invoked Providence, as the alternative of accident, to account for the "coincidences." Holmes's famous reply was, "There is a point of mental saturation, beyond which argument cannot be forced without breeding impatient, if not harsh feelings towards those who refuse to be convinced. If I have so far manifested neither, it is well to stop here, and leave the rest to those younger friends who may have more stomach for the dregs of a stale argument."

Charles D. Meigs should be remembered not only for his uncompromising opposition to great medical discoveries but also as an exponent of conservative obstetrics and as a contributor to the establishment of scientific obstetrics in America. He was a pioneer in the new era of obstetrics, even though he was proven to be completely wrong in so many instances. Medical progress is enhanced by vigorous opposition.

Hugh Lenox Hodge (1796-1873), professor of obstetrics at the University of Pennsylvania from 1835 to 1861, was a native of Philadelphia of Scottish descent. Like Meigs, he never failed to take issue with the authorities of his time whenever his personal observations and clinical experience were at

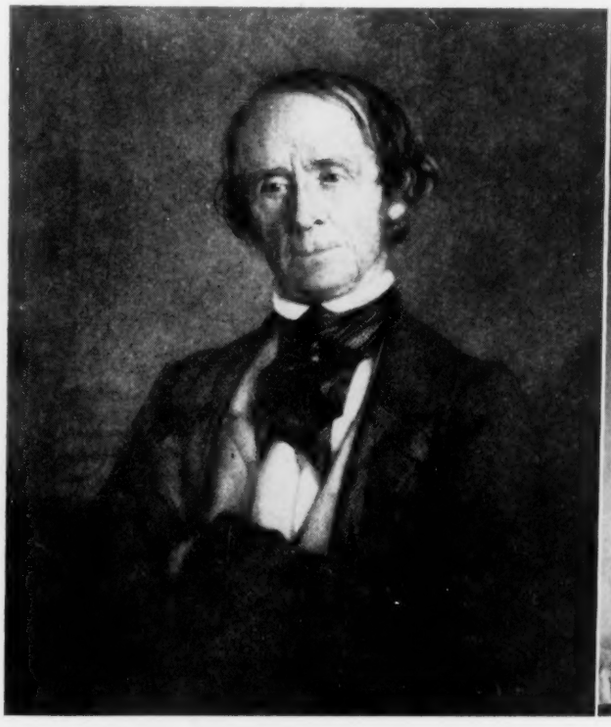

Fig. 1. Charles D. Meigs, 1792-1869 (Reproduced through the courtesy of the Jefferson Medical College Library)

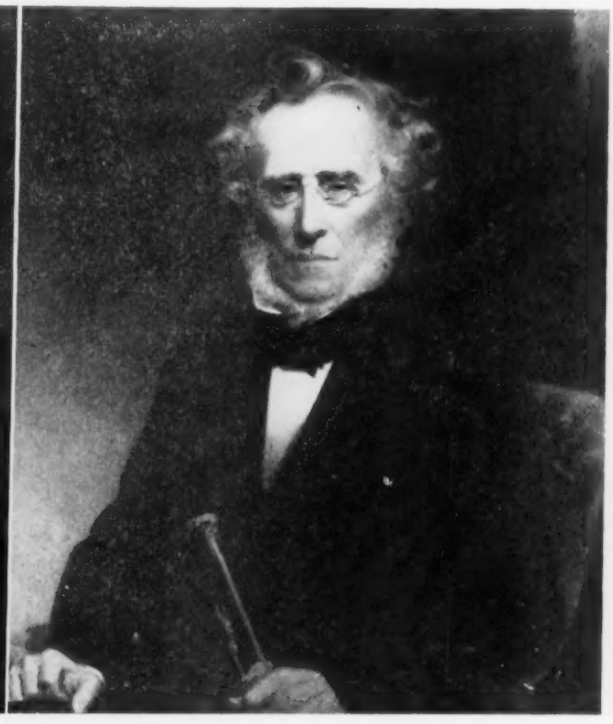

Fig. 2. Hugh Lenox Hodge, 1796-1873 (Reproduced through the courtesy of the University of Pennsylvania School of Medicine Library)

variance with their views.

Hodge was an excellent teacher and writer. Whitridge Williams considered his textbook, PRINCIPLES AND PRACTICE OF OBSTETRICS, published in 1864, as a masterpiece. Hodge also had considerable mechanical genius, and devised a placental forceps, long obstetric forceps, craniotomy scissors, and a pessary which is still popular. His pessary was constructed upon true mechanical principles—supporting the uterus by sustaining itself against the vaginal walls rather than against the rami of the pubis.

Hodge's later years were difficult because of failing vision. He was unable to read for the last twenty years of his life, and had to retire from teaching in 1863.

Hodge is recognized in obstetric history as a truly great man. He condemned symphysiotomy and held the middle road in regard to cesarean section. He advocated that all obstetric operations should be performed, as far as practicable, in accordance with the natural mode of delivery.

In 1871, the University of Pennsylvania bestowed upon him a high honor, the degree of Doctor of Laws. He richly deserved this tribute.

WALTER L. THOMAS, M.D.
Duke Hospital

* * * *

WALTER CHANNING (1786-1876)

Pioneer in Obstetric Anesthesia

Walter Channing, M.D., who began the use of ether in obstetrics a few months after the first public demonstration of ether anesthesia at the Massachusetts General Hospital in 1846, was one of three talented brothers, sons of William Channing, a lawyer, of Newport, Rhode Island. One brother, Edward Tyrrell Channing, became a teacher of English at Harvard and exerted his influence on such pupils as Emerson, Holmes, Sumner and Lowell. The second brother, William Ellery Channing, was the leader of Unitarianism. Walter Channing, the "medical Channing" ("My brother preaches and I practice"), entered Harvard College in 1804 with his cousin, Richard Henry Dana. He took part in the "Rebellion of 1807," and thus failed to graduate with his class, although he received his degree in 1867. For his M.D. he went to the University of Pennsylvania. After graduation in 1809, Channing spent three years abroad, principally in Edinburgh and London, before returning to Boston in 1812 to practice obstetrics. Harvard welcomed back her erring son by conferring upon him an M.D. degree, and in 1815, only three years later, made him the first professor of obstetrics in the Harvard Medical School. He served his school for nearly forty years; for thirty of them he acted as dean.

Channing made three distinct contributions to medical thought. As early as 1819, when a young man, he studied cases of delirium tremens and strongly supported the view, not prevalent at that time, that sudden withdrawal of alcohol would not result in delirium. He made a "diligent inquiry on this point" in the Boston almshouse, and published his notes in the old *New England Journal of Medicine and Surgery* for January, 1819. In 1842 he recorded the first description of so-called hemolytic anemia of pregnancy. He considered transfusion as a method of treatment, but, fearing some unknown incompatibility, dared not try it. That the idea even occurred to him in 1842 discloses his scientific curiosity.

Channing was soon to find an outlet for his inquiring mind in etherization[1]. Two cases of delivery under ether were reported in May, 1847. Later in the same year Channing had used ether six times, and by 1848, when his monograph, *Treatise on Etherization in Childbirth,* appeared he could report on its use in more than 500 cases. In a storm of controversy over the use of ether, or indeed any anesthetic in childbirth, Channing held a strong hand and played it with great skill.

A devout Unitarian, an ardent temperance reformer, a pacifist, he was a gay, vivacious and irresistible person, humorous and brilliant in conversation. For a pleasing insight into Channing as a traveler in Europe in 1852, with comments on puerperal fever and a discussion of the ether vs. chloroform controversy, any physician will enjoy his letters to a medical friend, published as *Professional Reminiscences of Foreign Travel.*

HENRY R. VIETS, M.D.
Boston, Mass.

1. Channing, Walter: (a) A Case of Inhalation of Ether in Instrumental Labor, Boston M. & S. J. 36:313-318 (May 19) 1847 (Note: The delivery was performed on May 7); (b) Inhalation of Ether in a Case of Laborious Labor, Boston M. & S. J. 36:335-337 (May 26) 1847; (c) Two Cases of Inhalation of Ether in Instrumental Labor, privately printed, Boston, 1847 (Note: Contains the first two papers); (d) Inhalation of Ether, Boston M. & S. J. 36:366 (June 2) 1847; (e) Cases of Inhalation of Ether in Labor, Boston M. & S. J. 36:415-419 (June 24) 1847; (f) Six Cases of Inhalation of Ether in Labor, Boston, White & Potter, 1847 (Note: Contains the first three papers, with alterations and additions).

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JULY, 1947

CENTENNIAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION

While the reckless use of superlatives is an American failing, it is safe to say that the meeting of the American Medical Association in Atlantic City, June 9-13, was the greatest medical meeting ever held in the world. The fact that 1947 marks the centennial anniversary of the founding of the American Medical Association was responsible for the unusual interest in this year's program. Atlantic City hotels and the huge auditorium which housed the exhibits and most of the section meetings were filled to capacity. Many doctors were unable to secure reservations.

For several days preceding the formal opening of the annual session, various special societies held meetings. On Sunday at 11 a.m. religious services were held in the Convention Hall. The speakers were Rev. Ralph Cooper Hutchison, Rabbi Joshua L. Liebman, and Monsignor Fulton J. Sheen. On Sunday afternoon the annual conference of state presidents and officers competed for attention with a conference of county society officers.

On Monday and Tuesday general scientific meetings were held in the ballroom of the Convention Hall, where advances in medicine and surgery were discussed by experts from this country and abroad. North Carolina was represented in the panel discussion on emergency surgery by Dr. Barnes Woodhall of Durham, whose subject was head injuries.

The high light of the opening session on Tuesday night was, of course, the address of Dr. Edward L. Bortz, delivered after he was installed as president of the American Medical Association. This address, which made a most favorable impression on his audience, was published in the *Journal of the American Medical Association* for June 14.

The House of Delegates was in session for four full days—from Monday to Thursday — with the exception of Wednesday morning. There was an enormous amount of work to be done, and progress was delayed by the large number of distinguished visitors who had to be introduced to the House, many of whom made addresses of varying length and intelligibility. Ordinarily the reports of the officers and of various special committees have been heard and most of the resolutions have been introduced before adjournment on Monday morning. This year, however, the House was exactly twenty-four hours behind schedule. It was Tuesday morning before resolutions were called for.

The most dramatic occurrence of the meeting came on Monday morning after Dr. Sensenich, chairman of the Board of Trustees, called for an executive session and read to it a long telegram from Senator Murray to President Harrison Shoulders. This message denounced the National Physicians

Committee for continuing its fight on compulsory health insurance, and abused the A.M.A. for not acceding to the demands of President Truman for a national health insurance act. The telegram was dated the day before it was delivered, and had been timed for release to the press immediately after it was read to the House of Delegates. Obviously the senator had planned the affair as propaganda for his latest version of the Wagner-Murray-Dingell bill. The trustees of the A.M.A. prepared for the press a dignified but scathing reply, which was unanimously approved by the House, and which gained more publicity than did Murray's blast.

In contrast to Murray's boorish message was President Truman's congratulatory telegram to the A.M.A., which was couched in elegant language, and was most cordial. This was read to the House of Delegates on Wednesday afternoon.

The most important committee to report was the one appointed more than a year before, on Amendments to the Constitution and By-Laws. Their revision, as amended since the December meeting of the House of Delegates, was presented, and a motion that it be acted upon at the next annual meeting was passed.

Notwithstanding the large number of members and guests present at this Centennial, the occasion was not complete without the genial presence of Olin West. It was the first meeting he had missed in so many years that it did not seem fitting to meet without him. President Edward Bortz paid him a graceful and well-deserved tribute in his presidential address, when he referred to him as "one of our nation's great medical statesmen . . . who for more than twenty-five years had been a loyal servant to the medical profession."

North Carolina was represented by one of its regular delegates, Dr. Charles Strosnider, and two alternates, Drs. Grady Dixon and Wingate Johnson. Dr. Strosnider was for the second time made a member of the important Reference Committee on Credentials, and Dr. Johnson was appointed chairman of the Reference Committee on General Practice.

THE GENERAL PRACTITIONER HAS HIS INNINGS

The progress made within the past few years toward providing a more secure status for the general practitioner is really remarkable. When it became evident that the emphasis on specialization was endangering the supply of general practitioners, the leaders of organized medicine began to take steps to halt and, if possible, reverse the trend. The Section on General Practice, which had held trial sessions since 1943, was made a permanent member of the A.M.A. family in 1946. In Chicago last December the House of Delegates passed two resolutions for the general practitioner's benefit. One was intended to encourage the inclusion of general practitioners on the staffs of hospitals, and to discourage the requirement of certification as a requisite for membership on a hospital staff. The second resolution gave the green light to a certification board for general practice, to be set up under the auspices of the Section on General Practice.

In the Atlantic City session this year, the Council on Medical Education and Hospitals issued a supplementary report disclaiming any intent to close the staffs of hospitals to general practitioners, and urging that general practice sections be organized in hospital staffs. In his address to the House, President Harrison Shoulders stated that the most urgent need in medicine is increasing the number and availability of general practitioners, and recommended a special committee to study the distribution of medical care, especially in terms of the general practitioner. President-Elect Bortz recommended an additional session for the general practitioner in connection with the midwinter session of the House of Delegates. He also suggested that this midwinter session be rotated from one area of the United States to another, instead of being held in Chicago every year. The recommendations of both the retiring and the incoming presidents were unanimously adopted.

The committee appointed from the Section on General Practice to consider the advisability of setting up a specialty board for general practice decided that the establishment of such a board should be postponed for the present, at least. Instead, an organi-

zation to be known as the "American Academy of General Practice" was formed, with Dr. Paul Davis of Akron, Ohio, as its president. Dr. Davis was chairman of the Section on General Practice this year. The Academy of General Practice is to be a national organization, presumably analogous to the College of Physicians or the College of Surgeons. One commendable feature which is embodied in its by-laws provides that membership "shall terminate at the end of three years, and to be eligible for reelection . . . a member must have maintained his high moral and professional character, and must have spent a minimum of one month during his three-year period in postgraduate training of a nature acceptable to the membership committee."

The objects and purposes of the organization are given as follows:

1. **To promote and maintain high standards of the general practice of Medicine and Surgery;**
2. **To encourage and assist in providing post-graduate study for general Practitioners in Medicine and Surgery, and to encourage and assist practicing physicians and surgeons to participate in such training;**
3. **To encourage and assist young men and women to prepare, qualify and establish themselves in general practice;**
4. **To protect the right of the General Practitioner to engage in Medical and Surgical procedures for which he is qualified by training and experience;**
5. **To advance medical science and private and public health.**

Qualifications for membership are set forth in the by-laws:

"To be eligible for general membership, the applicant must be a physician engaged in general practice. He must be of high moral and professional character. He must have graduated from a medical school approved by the American Medical Association. He must be duly licensed to practice in the state in which he practices, and must be a member of the local Medical Association of the area in which he practices. He must have had at least one year of rotating internship at an acceptable hospital or the equivalent in postgraduate training. He must have been engaged in the general practice of medicine for at least five years immediately preceding the date of his application for membership. He must have shown interest in continuing his medical advancement by engaging in some postgraduate educational activities during said five year period. Under unusual circumstances **exception** to the foregoing rules can be made by the unanimous vote of the membership committee. A general practitioner is defined as one who does not limit himself to one field of medicine."

It is to be hoped that this movement will not become in any sense a secession from the American Medical Association. Those who were most active in its formation say that it is not intended to be so interpreted, and cite the requirements of graduation from a school approved by the A.M.A., and of membership in the local medical society, as evidence that cooperation with the parent organization and not antagonism to it is the aim of the new group. They argue that the general practitioners are entitled to a society of their own, just as much as are the surgeons, the internists, the pediatricians, and other specialty groups. Certainly this argument seems valid, and the American Academy of General Practice should do much to supply the country with more and better family doctors. With this new national organization and with the newly envisioned midwinter session of the A.M.A.'s Section on General Practice, there is every reason for the general practitioner to feel that at last he is having his innings.

* * * *

EDITORIAL NOTES

A.M.A. Centennial

North Carolina was well represented at the centennial meeting, both in the section meetings and in the scientific exhibits. Among those presenting or discussing papers in the scientific sessions were Drs. Barnes Woodhall, W. C. Davison, Wallace Kernodle, Grant Taylor, Keith Grimson, and Eugene Stead of the Duke University School of Medicine; Drs. Hamilton McKay, Haynes Baird, and Kenneth Lynch, Jr., of Charlotte; and Drs. Weston M. Kelsey, George T. Harrell, Robert L. McMillan, and Charles R. Welfare of the Bowman Gray School of Medicine. In the scientific exhibits Drs. R. L. McMillan, J. P. Rousseau, J. C. Wiggins, Jr., and George Harrell represented the Bowman Gray School of Medicine, and Drs. Keith S. Grimson, G. J. Baylin, H. M. Taylor, F. H. Hesser, and R. W. Rundles, the Duke University School of Medicine.

The opening session on Tuesday night began with a musical program by the Philadelphia Festival Orchestra. As an encore, the orchestra played the "Iwo Jima March," composed by President-Elect Edward L. Bortz.

* * *

Dr. Guy Dain, chairman of the Council of the British Medical Association, brought "a birthday present" from the British Medical Association to the A.M.A. This was a gavel and block made of wood from the mulberry tree which grew in Charles Dickens' garden nearly a hundred years ago. The tree had to be taken down to make room for the British Medical Association House, which stands there now.

* * *

The weather during the first half of the week was delightful, but the last two days were torrid. The heat and the strain of four days' continuous session were beginning to tell on the delegates toward the last. Speaker Roy Fouts, however, kept his good humor throughout perhaps the most trying session the House ever held.

* * *

John Pratt and his associates of the Medical Services Foundation found that no entertainment had been arranged for the A.M.A.'s invited guests for Thursday. At noon Wednesday they secured a list of these guests and their wives, and arranged for a luncheon in their honor in the Belvedere Room of the Traymore Hotel at 12:30 on Thursday. An excellent program was hastily prepared, and handsome invitations were printed and delivered to "The Board of Trustees and Officers of the American Medical Association and their distinguished invited guests." This was an excellent public relations job. The Belvedere Room was filled to capacity, and our guests were quite favorably impressed by the affair.

* * *

The scientific exhibits were well worth all one's spare time. They often present in the most attractive form the concentrated essence of research extending over months or years.

LOGORRHEA: A PUBLIC MENACE

Recently we have witnessed in this country a flare-up of a very old disease which has been endemic in all parts of the world from the beginning of recorded history. This disease appeared in aggravated form in Hitler and his associates; it has apparently long been prevalent in Russia, if we are to judge by the emissaries sent from that country to discuss so-called peace terms with her allies; but nowhere has it reached such virulence as in the United States Senate. Huey Long's case has long been the most famous example, but his was a sporadic form of the disease. In its last appearance it has been epidemic: Senators Morse and Taylor have been most seriously affected, but Senator Pepper and a few others have also been stricken.

The disease is a loathsome one, and one of its most offensive characteristics is that the real victims are not those who are afflicted with the disease, but those who are afflicted with the patient. Only the exhibitionist is susceptible.

The disease under discussion is logorrhea (from the Greek *logos*, "word," and *rheein*, "to flow"). Dorland's MEDICAL DICTIONARY defines logorrhea as "excessive or abnormal volubility"; Webster's definition is "excessive and often incoherent talkativeness."

Those old fashioned enough to have read Tennyson may recall that in "The Brook" he tells a poetic tale of a girl whose father's logorrhea drove her lover away day after day, until a friend made the supreme sacrifice of listening to the old man long enough to give the sweethearts time to make up their quarrel. This fictional hero at least had the satisfaction of knowing that he was suffering in a good cause. The unhappy colleagues of Messrs. Taylor, Morse, Pepper *et al.* did not have such a reward for the torture they endured.

While the victim of logorrhea is for the most part merely a public nuisance, he may upon occasion become a public menace. It is unfortunate that the archaic rules under which the Senate functions permit a mere handful of men with leather lungs, tireless tongues, and endless effrontery to thwart the obvious will of the overwhelming majority of their colleagues, and of the entire country. This is too close to government by minority to suit the temper of America.

CASE REPORTS

CLINICOPATHOLOGIC CONFERENCE

BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

Presentation of Case

DR. FREDERICK R. TAYLOR: We are rightly told to consider common diseases before we do rare ones. As I read the protocol, I will try to discuss certain common things as they occur to my mind; if we can eliminate them, however, we must consider rare ones.

A 48-year-old automobile repair man entered the North Carolina Baptist Hospital January 15, 1947, and died just one week later. About a year before admission he began to note moderate generalized weakness and shortness of breath. Six months before admission a skin rash appeared overnight; it involved the whole body except the face, and was worst on the legs. The lesions first appeared as small, erythematous, pruritic papules, but later became pustular and crusted. Except on the legs they gradually improved with treatment, but there they ulcerated and crusted.

About two months before admission, the patient's local physician found his hemoglobin level 50 per cent. He was becoming weaker and more dyspneic. Parenteral liver and iron did no good. A few weeks before admission his hemoglobin was 39 per cent, his red cell count 1,830,000, the white cell count 7,300, hematocrit 17 (a practically normocytic anemia), color index 1.1, corrected sedimentation rate only very moderately elevated (12 mm. in an hour). Slight anisocytosis and poikilocytosis, and occasional basophilic stippling were noted. There was said to be no free hydrochloric acid in his stomach contents following histamine. Parenteral liver, iron, dilute hydrochloric acid, folic acid, and vitamin B all failed to benefit him.

The patient had had occasional epistaxis for many years, and during his present illness had it about every other day, sometimes severely. This could account for much of his anemia, but how can we account for the bleeding? We shall consider that point later. He had frequent mild frontal headaches during this illness. *Recently he had had increased postnasal drip;* he said this caused coughing, which was occasionally followed by nausea and vomiting. This postnasal drip may prove to be more than a mere incidental detail. The patient's wife said that his skin was yellow before the rash appeared. His soles felt "dead," as though he were walking on cushions.

Severe anemia with a slightly high color index, achlorhydria even with histamine, and paresthesias in the lower extremities make us think of pernicious anemia. Specific treatment for that disease failed, however, and a gastric analysis after admission showed plenty of free hydrochloric acid; therefore, we shall have to discard that diagnosis.

He had occasional colicky pain, and two loose, but not watery stools daily. Two months before admission his appetite failed, and after that he lost about 30 pounds. He had been in his present occupation for thirty years, and often had his hands in gasoline; only during the past year, however—that is, for about the duration of his symptoms—had the gasoline contained lead. It would be helpful to know how long before entering the hospital he stopped work.

The tetraethyl lead in gasoline is, of course, *organic* lead. This patient had a severe progressive anemia with some stippling of the red cells, but he lacked the other symptoms of tetraethyl lead poisoning, the outstanding and usually initial feature of which is maniacal excitement. There was no known exposure to inorganic lead other than the casual contacts with it that any automobile mechanic might have, working with batteries, and so forth. Furthermore, his colicky pains seem far too infrequent to suggest lead colic; there was no obstinate constipation or wrist drop; and the terminal clonic contractions of his arms were unlike the generalized convulsions of lead encephalopathy.

His father died of tuberculosis, and his mother probably died of the same disease during pregnancy.

The physical examination showed him to be well developed and well nourished, with pale skin and mucosa. A maculopapular eruption, complicated with bleeding scratch marks, was present over his arms, legs and body. His pupils and fundi were normal. The left nostril showed old blood. The thyroid, heart and lungs were not remarkable. His blood pressure was 140 systolic, 80 diastolic.

His liver was definitely enlarged (or pushed down), being three fingers' breadth below the costal margin. He had a reducible left inguinal hernia. His extremities were negative, except for the rash on the legs. The neurologic examination was essentially negative. No note is made of any objective disturbance of sensation in his soles. The rectal examination was negative.

The urine, which showed a very little albumin, a few white cells, and rare red cells, with no Bence-Jones protein, was hardly diagnostic of anything. There was a severe anemia, his hemoglobin being 7.3 Gm., and his red cell count 2,150,000. The hematocrit was 21 volumes per cent, which is again in keeping with a normocytic anemia. There were 1.8 per cent reticulocytes. This finding excludes the condition we call aplastic anemia, as there is no regeneration of red cells in it. The white cell and differential counts were normal except for 7 per cent eosinophils. As a number of skin conditions cause some eosinophilia, this may or may not be significant. The anisocytosis and poikilocytosis to be expected in an anemia were slight.

The serum albumin and globulin were not significantly disturbed. The Kahn test was negative, and the stool was negative for occult blood. This negative finding seems strange, for with all his nosebleeds, he must have swallowed a good deal of blood. The fasting stomach contents now showed 24 degrees of free hydrochloric acid and 50 of total.

X-rays of the skull, colon, and gallbladder were negative; a gastrointestinal series showed a deficiency pattern in the upper small bowel. I think this finding could well be attributed to his failure of appetite. *Fluoroscopy* of his chest was negative. It is unfortunate that there is no record of a film, since acute miliary tuberculosis may not show up on fluoroscopy.

While in the hospital, the patient developed furuncles of the thighs and legs. The sternal marrow showed slight hypoplasia of the erythrocytic series, again ruling out "aplastic" anemia. His temperature ranged between 98 and 101.2 F. Despite penicillin and a blood transfusion he became rapidly worse, and two days before his death complained of bilateral pain in his lower anterior chest on respiration. The next day a few moist rales were heard at the left base; a systolic murmur appeared at both the mitral and the aortic areas, and then he developed *clonic* contractions of his upper extremities. After the attack subsided, his blood pressure was 210 systolic, 110 diastolic, but soon fell to 150 systolic, 88 diastolic. I take this rise to be merely a transitory convulsive effect. Cheyne-Stokes respiration appeared the next day, and he died at 8:50 p.m.

Discussion

DR. ELBERT A. MACMILLAN (Neurologist): Dr. Taylor has asked what significance may be attached to the "clonic contractions." The description given is not sufficient to make possible either an etiologic or an anatomic neurologic diagnosis. The presence of a rather marked hypertension following the attack would suggest that there was some increase in intracranial pressure associated with the seizure.

DR. GEORGE T. HARRELL (Internist): The blood studies on this patient indicate that blood was being regenerated, though the anemia was not being corrected. Such a hematologic picture could be due to chronic blood loss (though the nosebleeds were the only source described) or to hemolysis. Lead intoxication may cause hemolysis. Industrial lead intoxication[1] is most frequently acquired by the inhalation of dust containing lead compounds. Lead intoxication usually leads to the release of young erythrocytes which are polychromatophilic or contain basophilic stipplings. Gastrointestinal symptoms, with abdominal cramps, occur very frequently. The neurologic signs are usually peripheral (neuritis) rather than central (encephalitis). Alterations in kidney function, and hence in the urine, are produced by the renal lesions. Since lead is excreted in the urine, the diagnosis can be readily proven by quantitative determinations on twenty-four hour urine collections.

DR. TAYLOR: One diagnosis I think I can make with dead certainty, and that is a left inguinal hernia. Unfortunately, however, this is a side issue which has nothing to do with the patient's death; it is what Dr. Richard C. Cabot used to call an "anatomical landmark."

The patient's illness lasted too long to be due to an acute pyogenic infection from the

1. Wampler, F. J.: Principles and Practice of Industrial Medicine, Baltimore, Williams and Wilkins Company, 1943, pp. 202-240.

start. The absence of chills and spiking temperature makes such a diagnosis even more unlikely. I have tried to exclude pernicious anemia, lead poisoning, and "aplastic anemia," whatever that is. The question of tuberculosis bothers me a bit. Could there have been some hidden focus that at the last exploded as an acute general miliary tuberculosis? This possibility seems unlikely to me, but I wish a chest film had been made. I see no evidence of an ordinary malignancy. I am no hematologist, but the differential count seems to exclude leukemia. Leukemia cutis may simulate many other conditions, but it is likely to involve the face; eventually the lesions become large, often coalescing and even producing a leonine facies almost suggestive of leprosy. Such characteristics are absent here.

Some common skin diseases, notably *scabies* and *pityriasis rosea*, spare the face. Since these are never fatal, however, and do not behave like this patient's rash, we may dismiss them even as coincident diseases. Apparently we are up against something unusual, without pathognomonic symptoms.

The rash does not resemble that of *mycosis fungoides*, with its tendency to develop ulcerating tumor masses. The eruption of *sarcoid* does not itch, and usually involves the face. Furthermore, this patient apparently had no lymph node involvement or deformity of the fingers, and no positive pulmonary findings. Since the cutaneous form of *periarteritis nodosa* usually shows hemorrhagic bullae sooner or later, I think it can be dismissed. *Disseminated lupus erythematosus* would almost certainly involve the face.

I believe we have a low-grade infection of some kind, perhaps affecting the bone marrow from the first, though with practically no effect on the white cells, later involving the skin, and finally the brain, with bilateral irritation of the motor centers controlling the upper extremities. It did not yield to penicillin. If we can rule out tuberculosis, I can think of no bacterial, viral, rickettsial, or protozoal disease that could produce such a picture. What, then, remains? Obviously, we must consider those mycoses which can become generalized. Here I am on admittedly unfamiliar ground.

Blastomycosis seems to be excluded by the lack of scarring and massive skin lesions. The nodules of *sporotrichosis* are absent, and the sudden appearance of the rash overnight seems to exclude that. In *coccidioidal granuloma*, and also in *toxoplasmosis*, when it becomes severe and generalized, we would expect more signs in the lungs than were found by physical examination or fluoroscopy.

Torulosis is to be considered seriously. It tends to affect the meninges and brain, and may affect the skin too. It often produces a "chronic meningitis." However, I can find nothing about nosebleeds or anemia in torulosis.

One fungus disease notoriously does affect the bone marrow, producing a severe progressive anemia, but having little effect on the white cells, except that eosinophilia may occur. That is *histoplasmosis*. A naso-oral type has been described, with single or multiple ulcerations in the nose or mouth, or both. This patient was subject to nosebleeds and complained toward the last of an increasing postnasal drip. This naso-oral type is often associated with cutaneous histoplasmosis.

My first guess is *histoplasmosis*, with *torulosis* as my second choice and some unusual type of *tuberculosis* as a very poor third. With any diagnosis whatever, I am unable to explain the fact that the rash spared the face, and the absence of pulmonary findings.

Anatomic Discussion

DR. W. C. THOMAS: At postmortem examination, the basic pathologic findings were noted in the kidneys. These organs were markedly contracted and weighed 75 Gm. each. The capsules stripped with difficulty and the surfaces were coarsely and irregularly granular. The architecture of the cut surfaces was obscured by the scarring. The pelves presented no abnormalities. Studies of the microscopic sections revealed hyalinization of many glomeruli. Other glomeruli showed adhesions between the visceral and parietal layers of the Bowman's capsules, which showed evidence of old proliferative activity. The interstitial tissue was infiltrated by mononuclear cells and lymphocytes.

This case is of interest from several points of view. It portrays the slowly developing picture of uremia, the chief presenting symptom of which was itching of the skin. The question of the relationship of tetraethyl

lead exposure and chronic glomerulonephritis can only be decided when complete clinical, experimental, and toxicologic information is available.

Anatomic diagnoses

Chronic glomerulonephritis

Uremia (excoriations of skin and clinical findings)

Closing Discussion

DR. WILLIAM A. WOLFF (Clinical Chemist): Dr. Taylor is quite correct in saying that we are up against something unusual, without pathognomonic symptoms. This is a rare disease—so rare that it has no generally accepted name, and so rare that it is almost unnoticed in medical literature. On seeing this type of patient, the clinician is startled and is very sceptical about the implication of his findings. The patient "feels well" and refuses to believe that he is suffering from a severe and fatal illness. There is almost nothing in the history or physical findings to suggest a serious pathologic process. The diagnosis is made *solely on laboratory findings*. The nonprotein nitrogen shows a marked elevation, sometimes 200 mg. per 100 cc. or more. The chlorides and total base are usually low. There is a marked acidosis, indicated by a low carbon dioxide combining power and a marked elevation of inorganic phosphates. A normocytic anemia, with red cell counts of 2,000,000-3,000,000, is usually found. The urine shows only a slight albuminuria, with occasional casts and a fixed low specific gravity. All the renal function tests show extensive impairment; the urea clearance is usually 10 to 20 per cent of normal.

We usually think of renal disease in terms of its complications—atherosclerosis, retinal changes, hypertension, cardiac insufficiency, edema, and gross albuminuria with hematuria. We fail to think of renal impairment apart from associated pathology. As a matter of fact, uncomplicated renal insufficiency is seldom seen by the physician. One of my associates at a large hospital in Philadelphia, in the course of twenty years, has seen between 30 and 40 cases of this type. In my own experience with blood chemical studies on some 200,000 hospital admissions, I have discovered 9 or 10 cases. I am confident that the incidence of uncomplicated renal insufficiency is much higher in the general population than these figures would indicate. The patient has no pain, "feels well," and therefore does not consult a physician or enter a hospital.

This type of renal insufficiency is the most interesting and the most neglected type of renal disease which I have encountered. An intensive study of this syndrome is well worth while.

DR. DAVID CAYER (Internist): Familiar diseases not uncommonly appear in altered guise. While we recognize that patients with chronic nephritis and renal failure may show marked gastrointestinal disturbances with nausea, vomiting and diarrhea, or anemias which fail to respond to usual forms of therapy, or even cutaneous changes secondary to uremia, the occurrence of these signs and symptoms without the other clinical evidences of hypertension, cardiac enlargement, eyeground changes, or urinary findings is most unusual.

It was somewhat unfair to Dr. Taylor to present him with this complicated problem and deliberately to omit the nonprotein nitrogen determination. We felt, however, that the omission of this laboratory finding would result in more discussion, and would show the case essentially as the diagnostic problem it presented to us. The diagnosis was suggested the day following the patient's hospital admission when he developed signs of air hunger, obvious acidosis, and increasing irritability suggestive of hypocalcemia and uremia. The additional laboratory data were then obtained and confirmed the clinical impression.

NORTON MEDICAL AWARD INVITES MANUSCRIPTS

W. W. Norton & Company are again offering the Norton Medical Award for book manuscripts written for the lay public by professional workers in the field of medicine. Terms of the Award have been slightly altered. The publishers now set no final closing date for the submission of manuscripts, which may be submitted at any time, the Award not being limited to any one year. The Norton Award offers $5000 as a guaranteed advance against royalties. Either complete manuscripts or detailed table of contents, together with one hundred pages of manuscript, may be submitted. A descriptive folder giving full details of the terms of the Award may be secured on request from the publishers, W. W. Norton & Co. Inc., 101 Fifth Avenue, New York 3, N. Y.

Books that have previously won Norton Medical Awards are **The Doctor's Job** by Carl Binger, M.D., **Doctors East, Doctors West** by Edward H. Hume, M.D., and **A Surgeon's Domain** by Bertram M. Bernheim, M.D., published this spring.

Psychosomatic Conference

Duke University School of Medicine

Medical History and Findings

Dr. H. King: A 26-year-old white single female was seen in consultation, at Dr. Magruder's request, regarding a history of tuberculosis. The patient, who had tuberculosis which had been arrested for some time, refused to believe that the disease was no longer active, and felt that she had a cavity in the left upper lobe.

The physical examination revealed a well developed, fairly well nourished white woman in no distress whatsoever. I could find nothing to suggest active tuberculosis. She had a rather profuse postnasal drip. The right chest was entirely clear, save for some compensatory over-aeration. The left chest demonstrated a definite respiratory lag. The left diaphragm was paralyzed from a permanent phrenic crush, and was elevated. Examination of the left lung revealed signs of a thickened pleura. There were also evidences of some contraction of the left lung, the mediastinum, trachea and heart being deviated to the left. The point of maximum impulse was felt in both the third and fourth interspaces; no significant murmurs were present. The peripheral vessels were clear. Examination of the abdomen was negative.

Fluoroscopic examination failed to demonstrate any signs of active tuberculosis. There was a definite shift of the mediastinum to the left. There was considerable calcium in the lungs, especially in the left upper lobe, and some fibrosis. The chest films showed numerous old scars, but no signs of fresh disease.

and the differential count was within normal

The patient's white cell count was 5,920, limits. Examinations of the sputum and gastric lavage revealed no acid-fast organisms. Serologic tests for syphilis were negative.

I talked with this patient for more than an hour, and explained fully the course of tuberculosis and the nature of her disease process at present. I do not feel, however, that she was yet willing to accept the fact that she has no active tuberculosis.

Her parting remarks were, "That may be, but something is causing my sore throat and vomiting. What is it?"

Psychiatric History and Findings

Dr. William Magruder: This 26-year-old, white, single girl was first seen in the Duke Psychiatric Outpatient Clinic on November 30, 1944, at the request of the Wilkes County Welfare Department. They requested psychiatric aid because, although all recent studies showed that the patient's tuberculosis was arrested, she would not be convinced of this fact and insisted on staying in the Wilkes County Sanatorium. She was admitted to Meyer Ward, the psychiatric division of Duke Hospital, on January 30, 1945.

The patient is the oldest of six children. The mother, who is living at the age of 44, had a fifth-grade education. Her only physical complaint is progressive rheumatism. When interviewed, she seemed quite concerned over her daughter's welfare and believed she still had active tuberculosis. She was perturbed because the patient was in a psychiatric division, and was quite sure that eventually we would send her to another sanatorium. The father is 53. He owns a 70-acre farm which barely provides a subsistence for his large family. The patient has one sister, aged 23 and single, who works in a hosiery mill and provides the patient with the niceties of life. Her four brothers, whose ages range from 21 to 11, are all at home. Two of them help their father with the farm, and one is still in school. The fourth brother, aged 17, recently acquired a backache and just sits around all day; he has not sought medical aid. The mother says that her children never got much schooling because the teacher didn't help them or encourage them. They never got along well with the teachers, and her husband let them stop school.

The home consists of two rooms and is quite crowded. The family has always had only the bare necessities, and at times not those. The siblings have felt their poverty keenly. There have been many frank and open quarrels between the parents, and the mother has left the father for periods of a few days on various occasions.

The patient's birth and early development were normal, and there were no early neurotic traits. She began school at the age of 6 and completed high school without failing, and with fair grades. She got on well with her teachers and schoolmates, except that she was often called the teacher's pet. It was

difficult for her to finish school, because she had few clothes and few books. She worked on the farm from an early age, doing menial work. She never complained or attempted to get out of any work. She had one remunerative job, which was working on Saturdays in a dry goods store. Just after graduation from high school, she went to live with an invalid aunt until the aunt died; shortly thereafter the patient was hospitalized.

The patient has had no heterosexual contacts. While in the sanatorium she met a young man who eventually asked her to marry him. Her reply was that if she got well, she would. She says, however, that she has no intention of marrying the man, but has just led him on. She corresponds with him freely.

In 1936 the patient began to lose weight and had blood-streaked sputum. In 1938 she had a massive hemoptysis and went to the hospital, where a diagnosis of tuberculosis, with cavitation in the left upper lobe, was made. After a few days in the local hospital, she was transferred to the Western North Carolina Sanatorium at Black Mountain. Pneumothorax was begun, but an effusion soon developed. She was kept on bed rest, and later in the same year a temporary phrenic crush was done. She remained in the Black Mountain Sanatorium for two years, and toward the end of that time was allowed to be up and was given work on the telephone switchboard. She stated that the work was too much, and that she had a complete recurrence of symptoms and massive hemoptysis. In her words, she was then sent to the fourth floor, "where they all die." In 1940 she was discharged to the "T.B. Hut" of Wilkes County. She remained there until her admission here in January.

According to the patient, the home was run by two laymen. The patients had to care for themselves or die. Rules were not enforced, and bed patients did not stay in bed. The patient said, "All of them died but me, and they sent me to Duke." Physicians seldom came to the "Hut," and sputum examination was rare. The patient described vividly the deaths of many of her friends; she was actually present during some of these deaths. Most important is the fact that nothing was done in the way of occupational therapy, and the patient said that she never heard a cheerful word during her sickness. Her parents came frequently, but they would stand in the corner and cry. She said that the doctor was cheerful in her presence, but that she would hear him discuss her and others outside the door, and always he would say, "Well, it's hopeless. They'll all die." In spite of the gloomy picture which is painted, the patient had a rather pleasurable existence. She acquired a radio and was provided with all the luxuries of a bed patient—bed jackets, housecoats, and many pairs of nice pajamas. She made many friends who have remained close, as is shown by her large correspondence.

When the patient was seen in the psychiatric outpatient clinic, the predominant picture was that of reactive depression; she cried a great deal. When first seen on Meyer Ward she was busy unpacking her belongings, which consisted of eight to ten pairs of pajamas, cosmetics, and various medicines. She insisted on getting into bed immediately. She kept a sputum cup, but produced only saliva. She complained of vomiting and of a sore throat which she thought was tuberculous. She remained in bed for approximately three days until her family could buy and send her clothes.

There was nothing very important in her mental status other than the mild paranoid trends. The patient was bitter toward every physician who ever treated her. Her intelligence was slightly below normal.

Daily interviews were held, which were unsatisfactory in regard to finding any psychodynamic factors, and which always ended in an attempt to convince her that her disease was inactive. She always replied, "Wait until I hemorrhage; you'll see." She attended occupational therapy classes in the morning and a special occupational therapy class in the afternoon. She was encouraged to take daily walks, and to go to campus movies, and to take part in other activities about the campus. She proved to be quite sociable, and was soon wrapped up in outside activities, although she declared that she was doing it at a risk. She was given small doses of insulin before each meal as an appetite stimulant. It worked well, and she made small gains in weight. Toward the end of her stay she became quite bitter toward me, probably because I was spending more time with another patient at that time. However, she has written two nice letters since.

She has an intelligence quotient of 82. A Minnesota Multiphasic Test was given, and the report showed that there is a possible lie factor and definite hysteria and depression factors. The paranoid quotient was also a little high.

The patient was discharged to her parents. I talked to her mother, who was by this time convinced that we were working on the right track. She has gotten along quite well at home and has taken steps to get into vocational rehabilitation offered by the state. She believes that her disease has been arrested and seems able to carry on a normal routine.

Discussion

DR. E. E. MENEFEE: I think that in every patient who has tuberculosis there is a psychiatric factor. It frequently falls to my lot to break the news to a person that he has tuberculosis. Most such patients go into a very definite depressive state for a few days. I think this is normal. Fortunately, most of them soon adjust themselves quite well with the help of the doctor, who emphasizes that tuberculosis is a curable disease.

Tuberculosis carries euphoria with it. I was impressed by this fact while serving in New York, where I saw cases so far advanced that even a layman could see that the prognosis was hopeless. Even patients who were in oxygen tents would invariably talk about "next year, when they would be well." Very few realized they would be dead in a few weeks.

I am glad that Dr. Smith is here today. It seems that there are two schools of thought regarding therapy of tuberculosis. I came from a place representing one extreme, and Dr. Smith's work represents another extreme. The therapy used in the sanatorium where I worked consisted of intensive bed rest. The importance of this was impressed upon the patient. Dr. Smith worked in a place where the patients were encouraged to get up as soon as possible. I have thought about these two schools in the past several years, and I think both of them have certain advantages. In upper state New York, where the patients are encouraged to get up early, it is rare to see any depression in a patient. In New York City, where they are very strict about bed rest, the patients are more likely to become depressed. We don't know which method cures more patients. But we do know that we should not leave the patient in a state of depression when his tuberculosis is arrested, nor should we leave him with a phobia about his disease. I have seen convalescent patients who are absolutely afraid of doing anything and fearful of assuming any normal life.

In this particular case, I wonder if the patient would not have had the same emotional reaction to any infectious, communicable disease.

DR. D. T. SMITH: This thing we call euphoria is the result of a set of circumstances. Tuberculous patients are maintained in a good state of nutrition and are certainly well rested. They can produce lots of sputum and not suffer. There is no nausea or vomiting. There is no reason for them to be depressed. They can't visualize big holes in their lungs, because they feel very well. Under such circumstances, I think euphoria is a normal reaction. The percentage of patients with a pessimistic outlook is much larger in other diseases.

DR. MENEFEE: Uncomplicated pulmonary tuberculosis is painless.

DR. SMITH: I agree with everything Dr. Menefee has said. This problem of the patient's reaction to his disease is most important. No doctor is really successful in handling tuberculous patients unless he is a practical psychiatrist, a rule-of-thumb psychiatrist on the basis of experience. When the disease is first diagnosed, the patient falls into a black depression. He feels that the whole world is shut off to him; it is just like looking out of a window at a beautiful future and having someone draw the curtain down. The length of time the patient stays in that depression depends on the patient and how the doctor talks to him. Sometimes it lasts a week, and it is not abnormal for it to last several months. What the doctor tells the patient should depend upon his prediction of the patient's reaction. To a sensitive person he would say, "You have a tiny little spot, and it is just a matter of a short time in a sanatorium." On the other hand, he often has to deal with a patient who is very cooperative for forty-eight hours and then wants to go back to work. The doctor then has to "scare the living daylights" out of him to get him to rest at all. Only a patient who has had tuberculosis knows the meaning of rest in bed. It means relaxing on the bed,

not just staying in bed.

In the case presented today, many unfortunate circumstances combined to produce the patient's reluctance to accept the fact that she was ready to begin normal activities again. At Black Mountain Sanatorium an effort was made to put her on the telephone switchboard. Ordinarily such encouragement is all that is necessary to make patients feel better. Unfortunately, she had hemoptysis, so that it became fixed in her mind that even light activity would cause hemorrhage and a relapse. To make matters worse, she was put with patients who were really dying. Then, when she finished her period in the sanatorium and her disease was arrested, there was no place for her to go.

Tuberculosis is characterized by a tendency to relapses. It is often the case that a person will have one or several relapses before he is cured. After a relapse, the patient is usually somewhat pessimistic about ever getting well.

The obvious thing to do for this patient is to rehabilitate her, to get her a job. She will get along all right as long as she has someone to consult. Usually a person such as this would consult a tuberculosis specialist. I see patients who have been well for ten years usually once a year.

You have accomplished a real miracle with this girl. She should have a job that would provide her with more luxuries than her disease did.

DR. M. H. GREENHILL: A great many tuberculous patients resist progress after getting out of the sanatorium, do they not?

DR. SMITH: Saranac is full of patients like that. If a man has compensation insurance, he never gets well if he is that kind of person. He describes symptoms, and symptoms cannot be x-rayed. Another type of patient, more often a woman than a man, just stays in Saranac Lake and spends the rest of her life there.

DR. MENEFEE: Thirty-five per cent of the population of Saranac Lake have tuberculosis. At Saranac Lake the whole place closes up from 12 to 3 p.m. When a tuberculous patient gets there he feels at home, for Saranac is run by and for people with tuberculosis. They do not let life interfere with tuberculosis, or tuberculosis interfere with life.

DR. H. DE JONG: May not the euphoria characteristic of tuberculosis be due to toxic elements acting on the brain?

DR. SMITH: This theory is well known, but has never been proved.

DR. GREENHILL: I will briefly summarize the conference in this way. When this patient came to Meyer Ward, some of us were rather alarmed by two symptoms which she showed—namely, the suspiciousness and the hypochondriacal trends. She would not listen to a doctor. For this reason we considered this case more of a psychiatric problem than is found in the average patient with tuberculosis. Everyone who handles tuberculous patients sees a whole range of reactions. We as psychiatrists are interested not only in the responses of the average patient, which have been pointed out by Dr. Menefee and Dr. Smith, but also in the wider emotional deviations. It may be helpful to think of these as reactions to chronic illness. The tuberculous patient reacts more or less like other patients with chronic illness. In certain cases in which the personality warps in response to chronic disease, I like to use the term *somatic compliance*. This means, in effect, that the personality completely complies to what goes on in the soma. The characteristics of somatic compliance include the following: (1) hypochondriacal tendencies, (2) aggressive paranoid trends, (3) exaggeration of pre-existing personality problems, (4) secondary gain, (5) extreme dependency reaction.

You will recognize that this patient has shown all of these characteristics. I imagine, from what I know about the history, that she has always been a dependent person. She was both her mother's and her teacher's pet, and always relied upon her mother for praise and recognition. She seems to have shown again and again the pattern of becoming hostile when she was thwarted from following out her dependent role. Aggressive behavior was used commonly in the inter-personal relationships within the home, and she adopted the same type of behavior when she could not follow out her desires. It seems that she has always tried to be a model child. She carried on through difficult, menial, and monotonous work under deprivation in her home because that pleased her mother; copied her invalid aunt after she had taken care of this aunt, because being model and dependent meant complete identification;

and, finally, became a tuberculous type when once she had the disease, subscribing wholeheartedly to the behavior that other tuberculous patients typically show and to what was demanded by the sanatoria. When she was not allowed to be dependent at the County Tuberculosis "Hut," she became hostile, as she did here when she was firmly but kindly pushed out of her invalid role. Of course, she undoubtedly got some secondary gain out of having tuberculosis, for facilities in all the medical institutions to which she was admitted were better than those in her own home.

Treatment here was essentially not difficult, although it might have been if her aggression had not been received properly. The first step was to push her gradually, but firmly, out of her invalid role into activities. She reacted to this attempt with marked hostility, which she was allowed to ventilate but which was not returned to her. She was allowed to be temporarily dependent upon a psychiatrist who could conduct her action into constructive channels. Her lifelong pattern of conforming in the most model fashion possible to the person upon whom she was dependent or to the environment in which she lived made the course simpler, because she became a good psychiatric patient on her way to recovery.

Although we can truly generalize about the emotional reaction of the tuberculous patient, the degree of emotional deviation or somatic compliance often depends on the lifelong behavior pattern of the patient.

New Remedy for Motion Sickness

A new prescription specialty called Companol for "motion sickness" was announced recently by Dr. Theodore G. Klumpp, president, Winthrop Chemical Company, Inc.

Dr. Klumpp declared that the new chemical compound was a further development of what was first used by the armed forces during the war and described it as "a logical preparation to combat sea, air, train and automobile sickness." The product has been stocked and thoroughly tried for clinical evaluation on major steamship and air lines. "More than 80 per cent of the motion sick travelers studied obtained relief," Dr. Klumpp said.

"It is a mixture of scopolomine, luminal and atrophine. Motion sickness results from excessive stimulation of the organ of balance located in the ear, in addition to apprehension, nervousness and fatigue incident to modern travel. Scopolomine and atrophine are known to produce the best clinical results in counteracting the physical effects of unaccustomed motion, while luminal, through its calming effect, allays the apprehension and excitement which contribute to sea and air sickness."

MEDICOLEGAL ABSTRACT

J. F. OWEN, M.D., LL.B.

RALEIGH

NARCOTICS: *Entrapment exists only when government agents induce or originate criminal intent of a defendant. Failure of druggist to fill prescription as written, and the fact that some prescriptions are not filled until after the arrest of the defendant are no defense against the charge of illegal dispensing of narcotic drugs.*

The defendant in this case, a practicing physician, was charged on several counts with illegal dispensing of opiates (morphine).

The records show that a government agent made several trips to the vicinity of the defendant doctor's office with a drug addict, a hired informer. The agent searched the informer before the visits, then gave him money and directed that he enter the doctor's office for the purpose of securing drugs. Each time the informer came out with a prescription for morphine and ephedrine, and usually the agent accompanied the addict to a drugstore, where the prescriptions were filled and the sale completed. Several prescriptions, however, were postdated and unfilled when the defendant was arrested. Upon his first visit the informer complained of asthmatic attacks, and the doctor made a physical examination at that time before ordering treatment; no subsequent examinations were made, however. It was not stated whether or not signs of asthma were present. It was brought out in the trial that the defendant failed to observe numerous hypodermic scars on the informer's arms. It was also in the records that the physician accused the informer of being an addict, but ordered the drug despite this fact. The doctor also cautioned the man not to have all the prescriptions filled by one druggist.

The defendant was found guilty in district court of the illegal sale of morphine, from which conviction he appealed to the circuit court of appeals. Among other exceptions the physician's lawyers alleged that illegal means were used in securing evidence (entrapment), that he was arrested before some of the prescriptions were filled, and that the

druggist failed to furnish the combination of drugs ordered. Ephedrine was not included when the prescriptions were filled. As to the question of entrapment, the court stated that this exists only when government agents induce or originate criminal intent of a defendant, and that there is none when criminal intent is already present and the agents merely afford the opportunity for the commission of a crime. Regarding the failure of the informer to have the prescriptions filled before the arrest of the defendant, the justices were of the opinion that criminal intent existed at the time of writing the prescription, and that the overt act committed placed in motion the crime which was afterward consummated. The court held that the failure on the part of the druggist to include the ephedrine as ordered was immaterial, as the intention was to secure morphine, which was the drug legally prohibited. The conviction of the lower court was affirmed.

Physicians who feel that it is incumbent upon them to furnish professional care for those suffering from drug addiction should take advantage of special provisions authorized by the Narcotic Bureau. This matter should be attended to by the physician personally, and copies of all communications, as well as affadavits of other physicians, should be kept safely filed for future reference. In cases of this kind it seems to be the intent of the law to prohibit the sale and distribution of opiates to habitual users of drugs solely for the purpose of continuation and not for actual therapy and rehabilitation. There are no restrictions of course against prescribing opiates during the course of a physician's practice if a disease process exists which requires such medication. (V. 149 Fed. Reporter, Second series, p. 29, U.S. Circuit Court of Appeals, Second Circuit, May, 1945)

Whenever a considerable number of tuberculosis deaths occur in the homes of the community, a serious source of tuberculosis infection exists and undermines other control measures. The general hospital has an important role in reducing this hazard. By expanding facilities for care of tuberculous patients, particularly for patients during the pre-sanatorium period, the latter institution will be able to utilize its facilities for all patients needing long term care, or palliative treatment. The general hospital can provide the diagnostic service and short term care required by the large number of persons in whom case-finding programs discover minimal tuberculosis. —Jacob Yerushalmy, M.D., Hospitals, Aug., 1946.

TUBERCULOSIS ABSTRACTS

A Review for Physicians

Issued Monthly by the National Tuberculosis Association

Vol. XX July, 1947 No. 7

PHYSICIANS have long observed that tuberculosis attacks the upper lobes of the lung by preference. A physiological explanation for this fact has been advanced. This hypothesis, if accepted generally, would put the standard treatment for tuberculosis —bed rest—on a sound scientific foundation, and mark a significant advance in our knowledge of infection and therapy in tuberculosis.

Apical Localization of Phthisis

No discussion of human tuberculosis and its management is complete or convincing unless it takes into account the fact that, in its inception and throughout much of its course, phthisis in adults is confined to the upper third of the lungs. Most members of white and many members of yellow racial groups are almost immune to tuberculosis except in this region. It is a remarkable fact that, even after the sputum and gastric washings contain bacilli consistently, pulmonary lesions for years may be limited to the upper third of the lungs.

Explanations of this curious predilection for the apex have been unsatisfactory. Those who have most consistently urged bed-rest for the treatment of phthisis seem unconcerned with the apical localization and its significance in the pathology and therapy of tuberculosis.

The relation of man's erect posture to the physiological functioning of the lung is usually ignored. Recently published data on blood pressures in the right ventricles of human hearts show that when a tall, long-chested individual is standing or sitting the pressure of the column of blood from the right ventricle to the apex of lungs is equal to or greater than the mean arterial pressure in the pulmonary arteries. Thus the flow of arterial blood will be greatly restricted or even absent at the apices of the lungs of most adults while they are in the erect posture. Nor at such times is tissue fluid or lymph produced by filtration in this area since it requires nearly 15 mm. Hg pressure to overcome the difference in osmotic pressure between the plasma and the pulmonary tissue fluid.

As a result of the decreased flow of blood addition of carbon dioxide to and removal of oxygen from alveoli in the apical region will be minimal. Tubercle bacilli reaching this tissue will be in an optimal atmosphere to support their metabolism although this is probably not an important factor in apical localization.

As long as the subject is sitting or standing, the pulmonary arteries can bring few antibodies to the apical region and removal of bacterial products by lymph, or dilution by diffusion into the blood will be almost nil. Only when the patient is recumbent will these tissues be protected against accumulation of toxic substances. Removal of bacteria to the lymph nodes will be suspended during most of the waking hours, as will the replacement of antibodies and of monocytes from the blood.

In view of these facts, it is not remarkable that active tuberculosis so regularly begins in this region, or that it shows a predilection for the tall, long-chested individual and the lively young people who get a minimum of sleep. Lack of rest is important in that it gives the bacilli the maximal number of hours of optimal growth conditions and the tissues the minimal number of hours when the neutralization and dilution of toxic products, and the supply

of antibodies and blood-borne phagocytes are as adequate at the apices as elsewhere in the lungs.

The great frequency of silicotic lesions at the apex also may be due to the fact that dust particles reaching this region will be removed by phagocytes less rapidly, and the injurious substances will be diluted and removed more slowly than in other parts of the lung.

This explanation of apical susceptibility is borne out by the statistics on the relationship of pulmonary tuberculosis and heart disease. Where the pulmonary circulation is depleted, as in cases of pulmonic stenosis, the incidence of active tuberculosis is high. Where there is pronounced mitral stenosis and the pulmonary circulation is engorged, cases of pulmonary tuberculosis are a rarity. This is not a matter of academic interest only. If the explanation given above is correct, obviously the ideal treatment of the early apical lesion is complete recumbency for most of the 24 hours. Clinicians who insist on "complete bed-rest" should realize that propping the patient up in bed for more than half an hour at a time interferes with the correct postural management.

So long as bed-rest remains an empirical and nonspecific form of therapy, it will be difficult to enforce it on intelligent people who feel well. An understandable, physiological explanation will make their cooperation more certain. To every adult with an apical lesion the hazards of the erect posture and the value of recumbency should be stressed. However, complete bed-rest, with its attendant discomfort and higher cost of nursing care, may not be superior to many hours of recumbency with brief periods out of bed.

Prolonged bed-rest will be better borne if numerous and gradually lengthening periods of sitting up or strolling about can be alternated with recumbency. A consideration of the pathogenesis of the apical and subapical lesions suggests that this may be sound practice in patients not prostrated by disease.

Apical Localization of Phthisis, William Dock, M.D., American Review of Tuberculosis, April, 1946.

* * * *

Postural Factors in Apical Tuberculosis

The theory that apical tuberculosis is due to postural ischemia accounts for its frequency in those young adults, studious or playful or both, who get little rest and are continuously erect 14 to 20 hours daily. If accepted, this theory also explains why bed-rest alone is effective in so many of these cases but of much less value in those with pulmonary basal lesions or tuberculosis of the kidney, epididymis or skin. When patients are propped up in bed to read, ischemia of the apex is not abolished. The effectiveness of bed-rest is thus greatly increased by outdoor exposure during the day, as the breezes and bright light discourage reading and promote appetite and sleep by stimulating the skin and tiring the eyes. Where patients are outdoors during the day they spend the maximal number of hours quiet and completely recumbent. However, it seems not impossible that brief intervals out of bed for meals and to facilitate nursing would not unfavorably affect the "cure" and make it less demoralizing and costly.

The new understanding of apical tuberculosis will make such therapy as complete recumbency easier for patients to accept. While awaiting specific antibiotics capable of controlling this disease, physicians will welcome a reasonable explanation for the pathogenesis of apical tuberculosis and for the therapeutic method which is still the main reliance in dealing with the commonest cause of prolonged disability and of death among young adults.

Editorial, Journal of the American Medical Association, July 13, 1946.

BULLETIN BOARD

Minutes of Executive Committee Meeting

April 13, 1947

The Executive Committee of the Medical Society of the State of North Carolina met in the Hotel Sir Walter, Raleigh, North Carolina, on Sunday, April 13, 1947. The meeting immediately followed a meeting of all the committees of the Society and was called to order at 3:45 p.m. by President Coppridge, who presided.

The following members were present:

Officers:

Dr. William M. Coppridge, Durham, president
Dr. George Erick Bell, Wilson, first vice president
Dr. James B. Bullitt, Chapel Hill, second vice president
Dr. Roscoe D. McMillan, Red Springs, secretary-treasurer

Councilors:

Dr. Donald B. Koonce, Wilmington, Third District
Dr. M. D. Hill, Raleigh, Sixth District
Dr. D. M. McIntosh, Sr., Old Fort, Tenth District

By proxy:

Dr. Frank A. Sharpe, Greensboro, president-elect (Proxy voted by Dr. Coppridge)
Dr. John Cotten Tayloe, Washington, Councilor, Second District (Proxy voted by Dr. Bell)

Secretary McMillan stated that Dr. Rachel Davis, chairman of the Advisory Committee to the Auxiliary, had brought up several inquiries from the president of the Auxiliary. The first concerned the advisability of the Auxiliary's meeting at another time and place, in view of the shortage of hotel accommodations. After some discussion, Dr. Bell moved that the Executive Committee express the desire that the Auxiliary continue to meet with the Medical Society. This motion was seconded and carried.

Secretary McMillan said that the next suggestion from the Auxiliary was that the Society members pay their wives' dues to the Auxiliary at the same time they pay their own annual dues. A motion to ask the Auxiliary to continue collecting its own dues was seconded and carried.

The last suggestion from the Auxiliary concerned a memorial to Dr. Paul McCain. After considerable discussion, Dr. Hill moved that a committee, with power to act, be appointed to confer with the Auxiliary on this matter. President Coppridge appointed Dr. Koonce, Dr. Matheson, and Dr. McIntosh as members of this committee.

The report of Dr. Bell's committee to secure an executive secretary was then discussed. This committee recommended that the Society employ Mr. Edmund S. Swindell, Jr., as executive secretary, at a salary of $4,800 per annum and with an allowance for travel to be fixed by a committee consisting of the incoming president, the secretary of the Society, and the chairman of the Finance Committee. Following some discussion, Dr. Koonce moved the adoption of the report, with the recommendation to the future officers that the office of secretary always be held by a member of the Society. Dr. Bullitt added an amendment specifying that the medical secretary be the immediate superior of the executive secretary. The amended motion was seconded and unanimously carried.

President Coppridge appointed Dr. Deryl Hart to serve on the committee to judge scientific exhibits, in the place of Dr. Howard Bradshaw, who was unable to attend the meeting at Virginia Beach.

A motion to adjourn was seconded and carried.

New Hanover County Medical Symposium

A Medical Symposium will be held at Wrightsville Beach, N. C., August 22, 1947, under the sponsorship of the New Hanover County Medical Society. Speakers will be:

Dr. Isaac A. Bigger, Professor of Surgery, Medical College of Virginia, Richmond.

Dr. Hobart A. Reimann, Magee Professor of the Principles and Practice of Medicine, Jefferson Medical College, Philadelphia.

Dr. John T. Williams, Assistant Professor of Gynecology, Harvard University, Boston.

Dr. Carl H. Smith, Assistant Professor of Clinical Pediatrics, Cornell University, New York.

Dr. Fred M. Hodges, Clinical Professor of Radiology, Medical College of Virginia, Richmond.

The meeting will last from 11:00 a.m. until 10:00 p.m., with intermissions for lunch, cocktails, and dinner. The entire program will be held at Lumina Pavilion. Headquarters and Registration Desk will be at the Ocean Terrace Hotel. A list of hotels and cottages appears below. Doctors who wish to attend the symposium but are unable to obtain accommodations should write the Housing Committee, Box 203, Wilmington, N. C.

Ocean Terrace Hotel	$3.00 and up, single (European)
Kitty Cottage	7.00 and up, single (American)
Landis Cottage	7.00 and up, single (American)
Hanover Cottage	6.00 and up, single (American)
Ocean Inn	6.50 and up, single (American)
Atlantic Inn	3.50 and up, single (European)

News Notes from the Duke University School of Medicine

Resigning his position as instructor in the Duke University School of Medicine, Dr. E. G. Goodman returned to Wilmington to undertake the practice of internal medicine, allergy and hematology on July 1.

* * * *

Three members of the Duke University School of Medicine faculty have been appointed as expert consultants to the Surgeon General of the War Department. Dr. J. Lamar Callaway, professor of dermatology and syphilology, serves as consultant in dermatology; Dr. Elbert L. Persons, assistant professor of medicine and director of student health for men, is consultant in medicine; and Dr. Barnes Woodhall, professor of neurosurgery, acts in the capacity of consultant in neurosurgery.

* * * *

Promotions

Dr. L. D. Baker—Professor of orthopaedics

Dr. J. L. Callaway—Professor of dermatology and syphilology

Dr. Robert Graves—Professor of neurology

Dr. Barnes Woodhall—Professor of neurosurgery

Dr. Philip Handler—Associate professor of biochemistry

Dr. J. S. Harris—Associate professor of pediatrics

Dr. B. Black-Schaffer—Assistant professor of pathology

Dr. D. G. Sharp—Assistant professor of biophysics in experimental surgery and biophysicist to Duke Hospital

Dr. J. T. Cuttino—Associate in pathology

Dr. Max Schiebel—Associate in surgery

New appointments

Dr. George Margolis—Associate in pathology

State Board of Medical Examiners

The Board of Medical Examiners will hold a meeting on July 31, 1947, at the Atlantic Beach Hotel, Morehead City, for the purpose of interviewing candidates for licensure by endorsement of credentials.

News Notes from the State Board of Health

Deaths from all leading causes, except cancer and accidents, showed an appreciable decline during the first quarter of 1947, according to reports received by the Vital Statistics Division of the North Carolina State Board of Health. There was not a single death reported as the result of malaria, scarlet fever, or typhoid fever. Cancer fatalities numbered 635 during January, February, and March as compared with 623 during the corresponding period of 1946, while accidents of all kinds resulted in 592 fatalities. against 539 the first quarter of the previous year.

The most noteworthy feature of vital statistics reports compiled so far this year is the unprecedented increase in the number of births. During the three months for which figures have been recorded with the State Board of Health 28,892 babies were born in North Carolina, as compared with 21,299 during the first quarter of 1946—a year which broke all previous records up to that time, with 100,595 live births.

Deaths from all causes in North Carolina totaled 7,715 during January, February and March, this year, which was a decline of 233 under the total for the corresponding period of 1946.

Catawba Valley Medical Society

The Catawba Valley Medical Society met at Lake Sylvia on June 19. Speakers on the program were Dr. Robert F. Bell, Dr. Boyce Griggs, Dr. J. F. Reinhardt, Dr. L. A. Crowell, Jr., and Dr. A. M. Cornwell.

Forsyth County Medical Society

Dr. Thomas W. Baker of Charlotte was guest speaker at the June meeting of the Forsyth County Medical Society, held in Winston-Salem on June 10.

Halifax County Medical Society

A film on "Nutrition" was shown at the regular monthly meeting of the Halifax County Medical Society, held in Roanoke Rapids on June 13.

Asheville Guidance Clinic

The Asheville Guidance Clinic will suspend activities for the summer. During this time patients should be referred to 3 Cumberland Circle, Asheville, telephone 5555-W.

News Notes

The Legion of Merit has been awarded to Col. John D. Yeagley, M.C., A.U.S., Winston-Salem, for his service as assistant director and later as director, Health and Sanitation Division, Institute of Inter-American Affairs, from February, 1943 to November, 1945.

Southern Medical Association

Mr. H. L. Williams of Birmingham has joined the headquarters staff of the Southern Medical Association as assistant to the secretary-manager, Mr. C. P. Loranz.

Eye, Ear, Nose and Throat Seminar

Correction

The announcement published last month concerning the Eye, Ear, Nose and Throat Seminar to be held at Hendersonville on September 15, 16, 17, and 18 erroneously stated that the first two days would be devoted to ophthalmology and the last two to otolaryngology. The sessions on otolaryngology will be held on September 15 and 16, and those on September 17 and 18 will be devoted to ophthalmology.

(BULLETIN BOARD CONTINUED ON PAGE 438)

AUXILIARY

At the Virginia Beach Meeting of the Auxiliary the membership reached the all-time high of 1004 paid members. This does not include our honorary members—those women who have been members for twenty years or more. These valued members swell our ranks considerably more. Our increased membership gives us a boost for the new year and a real enthusiasm for the work ahead.

The Executive Board for the year 1947-48 is as follows:

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Ninth District—Mrs. Alfred Kent, Jr., Granite Falls

Tenth District—Mrs. S. S. Cooley, Black Mountain

PHYSICIAN WANTED

The community of Farmer in Randolph County is in need of a physician. This is a thriving, thickly populated community within ten miles of a modern, 100-bed hospital. Any doctor interested in locating here should communicate with Miss Hope Hubbard, Farmer, N. C.

BOOK REVIEWS

Osler's Principles and Practice of Medicine. Edited by Henry A. Christian, A.M., M.D., LL.D., Sc.D., Hon. F.R.C.P. (Can.), F.A.C.P. Ed. 16. 1539 pages. New York: D. Appleton-Century Company, 1947.

Dr. Christian has brought to the writing of the sixteenth edition of the **Principles and Practice of Medicine** the same endowment that Sir William Osler brought to the writing of the first edition: unusual professional acumen sharpened by long clinical experience, and a lifetime of practice in imparting information to others. After more than fifty years, the latest edition of "Christian's Osler" remains the outstanding single-volume textbook of medicine by a single author. The reader is inevitably impressed by the author's wide range of medical knowledge, by his discriminating selection of salient points in the discussion of diseases, and by his clarity of description.

The sixteenth edition is a worthy successor to a long line of notable publications. It measures fully up to expectations.

Progress in Gynecology. Edited by Joe V. Meigs, M.D., Clinical Professor of Gynecology, Harvard Medical School; Chief of Vincent Memorial Hospital, the Gynecological Service of the Massachusetts General Hospital; and Somers H. Sturgis, M.D., Chief of the Vincent Memorial Hospital Laboratory; Assistant Surgeon, Massachusetts General Hospital. 552 pages. Price, $7.50. New York: Grune & Stratton, 1946.

The authors have collected authoritative papers on gynecologic subjects into a volume which permits anyone to bring himself up to date on the progress which has been made in the field of gynecology in recent years. More than fifty experts have prepared papers dealing with subjects representing their particular interest in reseach and clinical work. The book is divided into ten major divisions, with the following titles: Growth and Physiology, Diagnostic Methods, Functional Disorders, Interrelationship of Endocrine Glands, Sterility and Reproduction, Infections and Their Treatment, Benign Growths, Malignant Growths, Operative Technique, and Preoperative and Postoperative Care.

A few of the subject headings of note are "Congenital Abnormalities of the Female Reproductive Organs" by Emil Novak, "The Morphological and Endocrine Basis for Menstrual Bleeding" by J. E. Markee, "Transfusion with Particular Reference to the Rh Factor in Gynecology" by Louis K. Diamond, "Functional Excesses of Uterine Bleeding" by E. C. Hamblen, "Masculinization, Virilism, and Hirsutism" by J. P. Pratt, "Endometriosis" by Walter R. Holmes, "Pelvic Lymphadenectomy in the Treatment of Cervical Cancer" by Daniel G. Morton, and "Vaginal Hysterectomy" by A. D. Campbell.

A sufficient number of illustrations are included to clarify major points in the text.

This volume is carefully planned, and fulfills an important need of each of the special fields. The highly specialized material has been carefully handled, so that it is readily applicable to problems occurring in generalized and specialized practice. It can be recommended to medical men, surgeons, gynecologists, and medical students as a valuable addition to their library.

Diseases of the Chest: With Emphasis on X-Ray Diagnosis. By Eli H. Rubin, M.D., F.A.C.P., F.C.C.P., Attending Physician, Division of Pulmonary Diseases, Montefiore Hospital and Country Sanatorium, New York; Visiting Physician in Tuberculosis and Physician-in-charge, Chest Clinic, Morrisania City Hospital, New York. 685 pages, with 355 illustrations (24 plates in color). Price, $12.00. Philadelphia and London: W. B. Saunders Company, 1947.

The vast amount of material on diseases of the chest contained in this volume makes it an excellent reference book for the internist, surgeon, and chest clinician. For the radiologist it is invaluable because of the author's special emphasis on the roentgenologic findings in chest diseases.

The subject matter is presented in a logical and orderly manner. In the discussion of individual diseases, the pathology, physiology, clinical symptoms, physical findings, roentgenologic features, differential diagnosis, and accepted methods of treatment are thoroughly discussed and beautifully illustrated.

Accurate descriptions of the diseases are given, and the author's opinion of valuable therapeutic measures is outlined in the light of his own considerable experience. The many pertinent case reports and illustrations are well chosen. A complete bibliography is supplied for further reference on each disease. The book is printed on excellent paper with abundant illustrative material, beautifully reproduced.

The student, chest clinician, and roentgenologist will find this volume a ready source of accurate information to help solve the many difficult problems encountered in chest disease. Dr. Rubin and the publishers are to be congratulated on this splendid publication.

A Textbook of Medicine. Edited by Russell L. Cecil, A.B., M.D., Sc.D., Professor of Clinical Medicine, Cornell University Medical College; Consulting Physician, New York and Veterans' Hospitals; Visiting Physician, Bellevue Hospital, New York City. With assistance of Walsh McDermott, M.D., Associate Professor of Medicine, Cornell University Medical College. Associate Editor for Diseases of the Nervous System: Harold G. Wolff, M.D., Associate Professor of Neurology, Cornell University Medical College. 1730 pages, with 244 illustrations. Price, $10.00. Philadelphia and London: W. B. Saunders Company, 1947.

The fact that Cecil's textbook has reached its seventh revision since the first appeared only twenty years ago is proof of its popularity. The seventh edition retains the double-column format of the sixth, as well as other desirable features, such as the table of normal values for clinical examinations. Sixteen new subjects are discussed in this edition, and so much new material has been added that it contains 164 more pages than did the sixth edition.

With each successive edition, the wonder grows that the editor is able to round up articles from 162 contributors and see that each one is ready in time to meet the publisher's dead line. The feat was all the more remarkable this time because the task was begun in the midst of the war, when many of the authors were in service or working overtime at home. Nevertheless, the job has been well done, and the seventh edition is a worthy successor to its predecessors. Until the eighth edition comes out, it will probably be referred to more than any other medical book in existence.

Radical Surgery in Advanced Abdominal Cancer. By Alexander Brunschwig, M.D., Professor of Surgery, University of Chicago. 324 pages. Price, $7.50. Chicago: The University of Chicago Press, 1947.

This monograph gives a frank and detailed account of the personal experiences of the author in performing extensive operations for the extirpation of advanced abdominal cancer in 100 unselected patients. The nonsurgical contraindications to radical resection of abdominal cancer, and the supportive treatment for patients undergoing these operations are discussed. Separate chapters are then devoted to the operations performed for malignant processes arising primarily in the stomach, small and large intestines, pancreas, liver, extrahepatic bile ducts, adrenal glands, and pelvic viscera. These discussions necessarily deal extensively with the removal of other organs which were involved by the spread of the original malignant process.

All of the 100 cases reported in this monograph were well studied and well followed, and the operative mortality in the group was 34 per cent. Seventeen per cent of the entire number received no relief from the surgical operations performed, while 30 per cent did receive some palliation. The remaining group of 19 per cent represents those who survived for long periods following radical surgery. Of this group of 19 patients, 6 died of carcinoma after an average of thirty-three months, while 13 are still living and well, their average survival time being forty months.

In operating upon these patients, Dr. Brunschwig undertook these resections with a purposeful disregard of the ordinary concepts of operability in intra-abdominal cancer. For this reason, the book is thoroughly stimulating, for it will cause all thoughtful surgeons to pause and reconsider their present ideas of what is operable and what is inoperable in intra-abdominal malignancies. It is far beyond the scope of this brief review to discuss the factors which Dr. Brunschwig considered in determining whether or not a given lesion should be resected. Furthermore, it is irrelevant whether the reader agrees or disagrees with the author's lines of thought and procedure. The volume should be not only read but studied carefully by surgeons, for it introduces many new concepts and modifies others. All surgeons should take advantage of every available means to cure or relieve patients with cancer by surgical methods until that happy day arrives when we shall have biologic methods for the early detection and adequate control of this dread disease.

Textbook for Psychiatric Attendants. By Laura W. Fitzsimmons. 325 pages. Price, $3.50. New York: The Macmillan Company, 1947.

One of the most difficult problems in psychiatric training has been that of finding an adequate textbook for attendants. The present volume fills a long-felt need in presenting in a simple but intelligent fashion the basic essentials for psychiatric attendants. It begins with a brief section on the history of psychiatry and then leads the reader into a discussion of psychiatric nursing, stressing the basic points which a beginner must know. A discussion of hospital routines, precautions, feeding and first aid is followed by a chapter on ward housekeeping and personal hygiene. Special therapies are described, and how-to-do-it routines are included in each instance. Nursing care for special disorders is outlined and simple treatment procedures are presented in step-by-step listings. Finally, a chapter on definitions and explanations is given to round out a volume which will certainly find widespread usage in every psychiatric institution.

Postgraduate Obstetrics. By William F. Mengert, M.D., Professor and Chairman, Department of Obstetrics and Gynecology, Southwestern Medical College; Chairman, Obstetrics and Gynecology, Parkland Hospital, Dallas, Texas. 392 pages. Price, $5.00. New York: Paul B. Hoeber, Inc., 1947.

The cover calls this volume "a treasury of practical guidance for the family physician." Dr. Mengert points out in the preface that the physician in an average community delivers about eighteen hundred women at the annual rate of approximately fifty, and that he encounters relatively few of the severe complications of pregnancy. For this reason the space assigned a given subject is roughly in proportion to the frequency of its appearance. The methods of management and therapeutic measures suggested are based primarily upon the new routines of Parkland Hospital in Dallas and those of the clinics of the State University of Iowa, with which Dr. Mengert was previously associated.

The author has succeeded admirably in collecting and presenting a wealth of material in a clear and concise manner. A large, clear print and numerous subheadings make clinical application of the text easy for the general practitioner. Each of the major subjects is presented with a distinct paragraph on the etiology, clinical course and symptoms, diagnosis, and treatment. In addition, the management of the major complications is summarized in outline form in many chapters.

Some clinics would question the recommendation of as much as 5 or 6 minims of obstetric posterior pituitary extract for the medical induction of labor (page 167), and the use of the intrauterine douche for the control of puerperal hemorrhage.

An excellent index permits rapid reference to various clinical procedures. This book may be highly recommended for the general practitioner as well as for the medical student.

Office Endocrinology. By Robert B. Greenblatt, M.D., C. M., Professor of Endocrinology, University of Georgia School of Medicine; Director, Sex Endocrine Clinic, University Hospital, Augusta, Georgia. Ed. 3. 306 pages, with 71 figures. Price, $4.75. Springfield, Illinois: Charles C. Thomas, Publisher, 1947.

The author has attempted to condense the available information on the endocrinology of the male and female generative systems into a concise and practical book. Little theoretical material is included; instead Dr. Greenblatt makes a direct approach to the clinical symptoms commonly encountered, devoting little space to the diagnosis or differential diagnosis of the pathologic lesion.

He is more enthusiastic than most endocrinologists in recommending the use of endocrine preparations in the management of abnormal uterine bleeding, dysmenorrhea, and amenorrhea. He is specific in his recommendations for treatment of the various problems, including the exact dosage and schedule of treatment he considers proper.

His generous recommendation of androgen therapy in the treatment of dysmenorrhea, the climacteric, and abnormal bleeding, and even in some cases of nocturia and loss of libido, is in contrast to the cautious administration of androgens usually followed in the special endocrine clinics.

The general practitioner will receive much specific instruction concerning the management of various genital endocrine problems in the male and female from a study of this book. He should be very careful, however, in his review of the contraindications and cautions which Dr. Greenblatt includes.

BULLETIN BOARD

(CONTINUED FROM PAGE 435)

Baruch Committee on Physical Medicine

Increased opportunities in rehabilitation for the 23,000,000 Americans disabled by accident, disease, maladjustment and war, were forecast in the annual report of the Baruch Committee on Physical Medicine issued recently. The advancements in physical medicine and rehabilitation in the period covered by the report were termed of real significance in medical history by the committee, composed of leading physicians and medical educators.

One of the most significant items in the report was the announcement that the Advisory Board of Medical Specialties has approved the establishment of an American Board of Physical Medicine which will qualify physicians as specialists in this field of medical practice. Such specialists will be known as "physiatrists," a designation which stems from the Greek words "physis," referring to physical phenomena or agents, and "iatreia," referring to a physician or healer. The term, which was recommended by the committee, has been adopted by the Council on Physical Medicine of the American Medical Association, the American Congress of Physical Medicine, and the Society of Physical Medicine.

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Veterans Administration

At least 16,000,000 veterans will come out of World War II, according to the latest Veterans Administration estimate. VA said the potential veteran population will increase until the official termination of the war, which has not been declared yet.

By comparison, World War I produced 4,627,000 veterans; the Civil War, 1,849,000 Union Army veterans, and the Spanish-American War, 381,000 veterans. This makes a combined total of 6,857,000 for the three wars, or considerably less than one-half of the minimum estimated for World War II alone.

United States Public Health Service

Dr. R. R. Spencer has resigned as Chief of the National Cancer Institute of the U. S. Public Health Service. Dr. Leonard A. Scheele, formerly Assistant Chief of the Institute, succeeds Dr. Spencer.

Dr. A. C. Ivy has been appointed executive director of the National Cancer Advisory Council, succeeding Dr. George M. Smith, Professor Emeritus of Yale University, who resigned some months ago for reasons of health.

* * * *

"The Hospital Act and Your Community," a booklet authorized last year by the Hospital Survey and Construction Act, tells in simple terms what the Act means. It should be especially valuable to any group or community that wants to build a hospital but does not know how it goes about getting federal aid for construction. Single copies are available free on request to the U. S. Public Health Service, Washington 25, D. C. Larger quantities may be purchased at 10 cents a copy or $7.50 per hundred from the Superintendent of Documents, Government Printing Office, Washington 25, D. C.

American Heart Association

Dr. Arlie R. Barnes, Chairman of the Board of Governors of the Mayo Clinic, Rochester, Minn., has been elected president of the American Heart Association for the 1947-48 term. Dr. Tinsley R. Harrison, Professor of Medicine, Southwestern Medical College, Dallas, Texas, has been named president-elect. Other officers include Dr. Carl J. Wiggers, Cleveland, Ohio, vice president; Samuel Harrell, Indianapolis, treasurer; and Dr. Harry E. Ungerleider, New York City, secretary. Mr. Harrell is the first non-physician to serve as an officer of the American Heart Association.

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